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ON
DISEASES OF THE VERMIFORM APPENDIX

ON DISEASES

OF THE

VERMIFORM APPENDIX

WITH A CONSIDERATION OF THE SYMPTOMS AND TREATMENT OF THE RESULTING FORMS OF PERITONITIS

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A DISSERTATION PRESENTED TO THE UNIVERSITY OF OXFORD FOR THE DEGREE OF DOCTOR OF MEDICINE, IN APRIL 1894

BY

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DISEASES

OF THE

VERMIFORM APPENDIX

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CHAPTER I

INTRODUCTION

It has long been recognised that the parts lying in the right iliac fossa are above all other abdominal structures liable to an inflammatory affection, which is generally marked by the appearance of a tumour in this region, and is sometimes followed by the formation of an abscess. This affection—localised peritonitis, as we now know it, "iliac phlegmon," as it was termed at the beginning of the century—is seldom fatal, and consequently the post-mortem observations which are necessary to explain its site and origin are to a great extent wanting.

For the first forty years of this century all these localised inflammatory affections in this region were attributed to disease of the cæcum. And although for a hundred years or more it has been known that disease of the vermiform appendix is capable of exciting an inflammation of the whole peritoneum, yet the theory of a cæcal origin for all local inflammation in the right iliac fossa has been unquestioned until recent times.

The cæcal theory has, however, recently been the subject of gradual modification, and it is now nearly if not quite extinct.

The vermiform appendix is a part of the intestinal tract, which is insignificant in point of size, and probably devoid of function. Disease of its mucous membrane can be shown to be of frequent occurrence, but this is in itself of no importance, and it is not as a rule attended with recognisable symptoms. Such disease, however, is wont to light up an inflammation of the peritoneum, sometimes of the whole peritoneal membrane, more commonly of that part of it only which is contained in the right iliac fossa. From the high rate of mortality accompanying the one case, and the widespread suffering entailed by the other, the appendix acquires a human interest which is wholly disproportionate to its physiological function.

Although the appendix has now obtained a tardy recognition as the chief factor in the production of peritonitis in the right side of the abdomen, yet the part played by it appears to be still underestimated.

Owing to the lack of post-mortem opportunities there is not in existence any clear and systematic account either of the changes in the appendix or of the modes in which peritonitis arises therefrom. Moreover, the clinical features of the disease are hardly presented in our text-books with the accuracy and minuteness that would seem to be demanded by a common morbid condition of such danger and difficulty. But a matter of higher importance is the great diversity of opinion which prevails as to the propriety and possibility of operative treatment. So soon as the cæcal origin of these cases of peritonitis became discredited, and the appendix was recognised as the source of the evil, the then universal method of treatment was at once exposed to doubt. The idea arose among American surgeons that the true treatment of the disease lay in a prompt excision of the appendix. Though their example has not been without influence in England, still the average practice in the two countries exhibits a wide divergence, and perhaps the national proclivities are discernible in the character of their respective views. There is a pressing need for a definite and uniform plan of treatment both in regard to this question of an early excision of the diseased organ and in regard to the general surgical management of all the stages and degrees of appendicular peritonitis. In spite of much valuable work bestowed on the subject in all countries during the last few years, it is clear that the evidence on which a decision may be made is still insufficient, and the matter remains in confusion.

It is on these grounds that a further contribution drawn from the records of a large general hospital may be justified.

NOMENCLATURE

Doubtless the obscurity and vagueness of some of the views that are expressed result in some measure from the babel of names that have been applied to the disease, and before entering upon a discussion of the subject it is necessary to define the terms that will be used.

The condition has received many names which are more or less expressive of the current idea as to its causation. It has at various times been called "iliac phlegmon," "typhlitis," "perityphlitis," "paratyphlitis," "cæcitis," "pericæcitis," "paracæcitis," "appendicitis," "periappendicitis," and "ecphyaditis."

Granting for the moment that the appendix is in all cases the source of the evil, and that the changes in the appendix are at bottom of an inflammatory nature, the word "appendicitis" becomes at once unavoidable as a term expressing the pathological process, indefensible though it be from an etymological point of view. This term has already been widely accepted, and it is now far too firmly established to allow of the intrusion of such a stranger as "ecphyaditis," however pure of origin it may be. It does not seem right, however, to use the word "appendicitis" in a clinical sense, inasmuch as it is probable that any symptoms resulting from it are due solely to the involvement of the peritoneum, which is not truly a part of the appendix. Consequently for clinical purposes the best and simplest plan is to speak of a "peritonitis," general or local, as the case may be, or, if there should be any ambiguity as to its origin, to define it as an "appendicular peritonitis."

As regards the "local appendicular peritonitis," the old term of "perityphlitis" seems a simple synonym, though less exact. Except in the cases of unusually placed and unusually long appendices, any inflammation around the appendix must almost certainly involve the peritoneal surface of the cæcum. Consequently the term is intelligible and approximately correct, if it be taken in the sense, in which it will be here used, of an appendicular peritonitis localised in the neighbourhood of the cæcum. The term, moreover, has been sanctified by long usage, and it is quite as justifiable as the generalised use of such a word as "cirrhosis."

The words "typhlitis," "cæcitis," are of course inexcusable, save in the mouth of one who believes in an inflammation of the cæcum.

At any rate the meaning of the three terms, which alone will here be used, is clear enough—viz., "appendicitis" as a pathological process, and "general peritonitis" and "local peritonitis" (or perityphlitis) as denoting clinical forms of disease.

CHAPTER II

Historical Review.—Bichat, Laennec, Ferrall, Mestivier, Dupuytren, Goldbeck, Copland, Burne, Albers, Grisolle, Villermey, Voltz, Rokitansky, Lewis, Wister, With, Fitz, Bristowe, Fagge, Osler.

Examination of the Ancient Cæcal Theory.—Perforating ulceration of the Cæcum, non-perforating ulceration, catarrhal inflammation.

Evidence on which the Appendicular Theory rests.

HISTORICAL REVIEW

In the eighteenth century not only did the appendix go unmentioned, but the peritoneum itself was not recognised as a continuous structure distinct from the organs which it invests. It was known to cover the various abdominal viscera and to line the abdominal wall, but it was not appreciated as being a single membranous structure, subject to its own specific diseases and pre-eminently liable to inflammation. Its recognition was due to Bichat's *Treatise on Membranes*, published in 1800. Still less had "peritonitis" any place in the field of practical medicine. Diseases were at this time recognised and classified by alteration of function alone. The less important the function of an organ, or rather the less obtrusive the function, the greater the mystery that surrounded its diseases.

Bichat.

This period, however, marked the beginning of the fall of tradition. Study of signs and symptoms alone, far as it had carried mankind in the knowledge of disease, was giving place to routine observation of morbid anatomy and the results of disease, and the centre of thought was shifting from bedside to post-mortem room.

Laennec.

One of the earliest fruits of this revolution was the appearance of Laennec's *Hist. d'Infl. du Péritoine* in 1803, and from this time forth the peritoneum began to assume importance. It was long, however, before its connection with disease in the right iliac fossa became established.

In the literature of the beginning of this century, descriptions of "phlegmon of the iliac fossa," as it was then called, are abundant, its symptoms and course being set forth with great accuracy. Few writers, however, ventured upon an explanation of its origin, or it might be that to a perfect clinical picture would be appended an account of an autopsy, in which no mention, positive or negative, is made of the appendix, and it was in fact never examined.

Ferrall.

One of the earliest and most important contributions to the subject was a paper by Ferrall[1] on "Phlegmonous Tumours in the Right Iliac Region," published in 1831, though written some years before this. We find here that the cæcum is held without question to be the organ primarily diseased, that the appendix is not considered, and that the peritoneum still plays but a small part as compared with the cellular tissue behind the cæcum. He takes the indurated mass so commonly found in the right iliac fossa as his standpoint, and concludes that it may be of three kinds: (1) a feculent tumour or distended cæcum (and herein he disagrees with Menière, who thought the tumour always inflammatory); (2) a malignant tumour of the cæcum; (3) a true phlegmonous tumour—*i.e.*, an inflammation of the cellular tissue behind the cæcum resulting either from irritation of the cæcal mucous membrane or from ulceration and perforation of the cæcal wall. He dismisses the subject of the peritoneum by adding that in most phlegmonous cases there is a certain degree of thickening of the peritoneal coat of the intestine.

There can be no doubt from his clear descriptions that Ferrall is here dealing with "perityphlitis," or the local form of peritonitis. As an example may be taken the case of a patient whom he saw five days after the sudden onset of abdominal pain, nausea, and constipation. "The abdomen was generally full, but yielded to pressure everywhere except in the right iliac fossa, where over a hand-breadth there was a good deal of tension and tenderness; the tongue was coated, the skin hot and dry, the pulse 90 per minute; the patient was lying on his back with the knees drawn up. An enema was given, and some scybala came away; the general fulness of the abdomen became less, while that of the right iliac fossa was now perceptible to the eye. Calomel 1½ gr. was then given every four hours, until mercurialisation was established in six days. The mass in the right iliac fossa slowly disappeared, but for a week or ten days a small knot could be distinctly felt on careful examination."

Mestivier.

Now as far back as the year 1759 Mestivier[2] had recorded a case of perforation of the vermiform appendix. And it seems curious that Ferrall had no idea that these milder phlegmonous tumours could have a similar origin. That the appendix could be the cause of the inflammation in any of the cases which he narrates was obviously never in his mind. His account of an autopsy is instructive on this point: "In the body of a boy who died after an illness of twenty-six days there was lymph all over the peritoneum, but on the right side six ounces of dark-brown serous fluid; the cæcum was covered with lymph anteriorly, and much thickened in all its circumference. In it the mucous coat was thickened and pulpy, but not easily scraped away; the colour was deeper as it reached the ileocæcal valve; the mucous membrane at this aperture projected considerably all round, and the opening was much contracted." He concludes by saying that the affection of the cæcum was the primary disease. The vermiform appendix was not examined, but it is not presumptuous at the present time to say that, had it been examined, it would have been found to be the true origin of the mischief.

Dupuytren.

We find the same views expressed by Dupuytren[3] in 1833, and it is indeed due to the name of the great surgeon that the cæcal theory of perityphlitis was so generally accepted, and was handed down to us as an unquestioned item in our inherited knowledge. Fifty years went by before this doctrine was definitely contested by any one. The vermiform appendix does not enter his view; in his account of an autopsy it is not even mentioned. We read of irritation of the mucous membrane of the cæcum, the frequency of which is explained by the position of that organ being such that foreign bodies are readily lodged in it, and the fæcal contents tarry in it under the influence of gravity. He speaks of inflammation propagated thence to the retrocæcal tissue, and of the cases where the peritoneum is also attacked, which he says are happily rare.

Dupuytren's explanation, erroneous as we now think, can easily be understood. Inflammation in the right iliac fossa does not commonly prove fatal, and when death does occur the parts in the fossa are so embedded in pus and thick lymph that their unravelling is a matter of considerable difficulty. The appendix readily escapes attention, and is indeed at times very difficult to find. Again, the intense inflammation of the peritoneal coat of the cæcum cannot but have a secondary effect upon the subjacent mucous membrane, which is manifested by hyperæmia and catarrh apt to mislead the observer; and when a local abscess is formed around the appendix, it not uncommonly perforates the wall of the cæcum, and from this again error would be likely to arise.

Goldbeck.

Goldbeck[4] in 1830 took the same view, notwithstanding the fact that he himself records a case of perforation of the appendix.

Copland.

In an article on Diseases of the Cæcum in his *Dictionary of Practical Medicine* (1834) Copland still further exalts the position of the cæcum as a source of illness. He writes with great minuteness of "disordered functions" of that part of the intestine, attributing to them many symptoms, some of great severity.

Under "inflammation of the cæcum" he describes with accuracy what we now know as perityphlitis, but like his predecessors he is of opinion that when the inflammation spreads beyond or outside the cæcum it is the retrocæcal cellular tissue, and not the peritoneum, which is affected. He shows however a great advance on the pathology of Dupuytren, for he recognises that the appendix can be the primary cause of severe trouble in this region. He speaks of its sphacelation as a result of the presence of foreign bodies in it with consecutive fatal peritonitis.

Burne. John Burne[5] in 1837 came nearer to the truth. Like Copland, he devotes a large part of his article to inflammation of the cæcum, giving us an insight into the great importance that was attached to this part of the intestine at that time. But the great merit of his work lies in the originality of his views with regard to the appendix, which he endeavours to separate clinically from the cæcum. He speaks of "ulceration of the appendix" set up by the presence of foreign bodies, such as raisin stones, cherry stones, and concretions, and he is familiar with the results. "So long as ulceration is limited to the mucous membrane it is of little consequence, but immediately the peritoneum is perforated inflammation ensues: then there is general peritonitis, or local peritonitis with abscess." So near the truth was he that two years later[6] he published a paper in which he departs still further from tradition by minimising the importance of the cæcum. He casts doubt on the alleged frequency of its perforation, and states his belief that all Dupuytren's cases were due to disease of the appendix, a belief in which every one must share.

Albers. A paper by Albers[7] in 1838 marks a step in the same direction. He fully recognised the possibility of disease in this region taking origin in the appendix, but gave it a subsidiary place beside the cæcum. If he found an abscess cavity with a perforation of the appendix, he was inclined to regard the perforation as a secondary occurrence.

The authority of Dupuytren was so strong in the schools of France that little or no originality of thought on this question made its appearance in the succeeding generation. In 1839 Grisolle[8] takes up the tale of the cæcum, though he frequently alludes to fatal cases of gangrene of the appendix. He has the great credit however of fully recognising that if an abscess forms it may be difficult to detect fluctuation; and he maintains in clear language that such an abscess must be opened, and must not be allowed to discharge into the bowel. In 1840 Villermey[9] published some cases of rapidly fatal inflammation and gangrene of the appendix.

Grisolle.

Villermey.

Every few years now brought fresh information on the subject of the appendix, while the cæcum fell more and more into the background; and this change is directly related to the growing habit of making post-mortem examinations.

Voltz. Thus in 1843 an excellent paper by Voltz[10] appeared, entitled "Ulceration and perforation of the appendix occasioned by foreign bodies." Here we find a series of cases both of general and local peritonitis, all of which he attributes to disease of the appendix, and with some relief we hear nothing of the cæcum or retrocæcal tissue. But this paper is confessedly a retrospective one. It is only on looking back on these cases, and maturely considering them in the light of subsequent experience, that he is led to the conclusion that the appendix was in all of them at fault. And though in many of them an autopsy was obtained, the appendix was in some not examined at all; and in one case of general peritonitis, though it was examined and found to be perforated, he had not at the time the curiosity to open it up.

Rokitansky. Rokitansky's article in his *Pathological Anatomy* (vol. ii. 1843) was a noteworthy addition to the current knowledge of diseases of the appendix. He describes a catarrhal inflammation resulting from the presence of fæcal matters and foreign bodies, which may become chronic or pass into ulceration. Herein is a great advance, for all previous writers had considered only such violent affections as gangrene or perforation. He further shows how from subsequent perforation a general peritonitis does not necessarily follow, inasmuch as the previous irritation has in most cases induced adhesion of the neighbouring peritoneal folds. But with this very accurate account, which is probably the outcome of his own experience, there is an odd mixture of tradition from which he cannot break loose. We read of the frequency of catarrhal inflammation of the cæcum, of resulting ulceration, and of perforation of its posterior wall, and inflammation of the retrocæcal tissue, which is Dupuytren's view again in all its crudity.

Lewis. A paper by Lewis[11] in 1856 takes us another step towards appreciation of the appendix. He collects a large number of cases where a fæcal concretion or a foreign body has originated such disease in it as to lead to general or local peritonitis by means of perforation. As a result of perforation he considers that general peritonitis is always speedily fatal, but that previous adhesions may circumscribe the discharge consequent upon disintegration of the appendix, thus postponing for a longer or shorter time its fatal termination. But he did not suspect that similar conditions of the appendix could result from a catarrhal inflammation of its mucous membrane, as well as from the presence of a concretion. In fact, if he found a diseased appendix without a concretion, he took it for granted that the latter had been broken up and discharged. Moreover, he saw no connection between the appendix and the milder degrees of inflammation in the right iliac region, which in his opinion had a purely cæcal origin. In fact, his view may be summarised by saying that if the inflammation was violent and fatal the disease originated in the appendix, but if it was mild and not dangerous (even though suppuration might occur) the cæcum was the seat of the original mischief. It must be allowed that this is the position still held by some authorities.

The appendix received careful study by Wister[12] in 1858. *Wister.* He gives an interesting case of local pelvic abscess resulting from perforation of the tip by a concretion, of which further mention will be made.

From this date onwards a vast number of isolated communications have appeared in current literature detailing cases of local or general peritonitis due to disease of the appendix; while with regard to the cæcum there is a profound silence, until in 1880 With[13] of Copenhagen was the first to state his *With.* entire disbelief in the occurrence of a peritonitis due to disease of the cæcum.

Finally appeared the exhaustive article of Fitz[14] in 1886. *Fitz.* Full of facts relating to the appendix and its diseases, drawn from all sources, it attracted great attention, and served to put the whole matter on a clear basis. The immense frequency of appendicular disease was at once placed beyond question. But even here the cæcum was still in view. It is true that he showed the extreme rarity of a primary perforating inflammation of the cæcum, but he described the symptoms of a "stercoral cæcitis" as an important condition, with which the perforating appendicitis may be confounded.

The same view appears in most of our text-books. Bristowe *Bristowe.* speaks of "typhlitis" and "perityphlitis," using the terms in their strict sense. He places the cæcum and the appendix on the same level as regards their power of exciting inflammation of the surrounding tissues—"inflammation of the cæcum or its vermiform appendage involves either by perforation or by simple extension the connective tissue of the iliac fossa or the peritoneal cavity." As regards the cæcum, he attributes ulcerative perforation to the extension of tubercular, typhoid, or dysenteric ulcers; to simple but extreme distension of the cæcum; to the fretting of its surface by accumulated fæcal contents; to the mechanical effects of bristles, pins, or bits of bone, which have been accidentally swallowed, or to the lodgment of intestinal concretions. As regards the appendix, he considers that a concretion is the usual cause of perforation.

Fagge, however, quoting Wilks, expressed the opinion that *Fagge.* the primary condition in both the affections known as typhlitis and perityphlitis is disease of the appendix and not of the cæcum. He thought that the cause of the inflammation of the appendix is generally the presence of a concretion, less commonly of a foreign body; that sometimes the ulceration is tubercular, but that in some cases no exciting cause can be found, unless it may be possibly a distension of its cavity. He did not, it will be noticed, lay much stress on the essentially peritonitic nature of the disease, for he attributed "the ill-defined rounded swelling, extending upwards from the iliac region, in part to the thickened coats of the affected portion of the bowel, in much greater part to the accumulation of fæcal matter within it."

Osler. Finally, in our most modern text-book, that of Osler (1892), the whole subject receives most rational treatment. All the forms and degrees of peritonitis arising from disease of the appendix are here for the first time brought together under one head. He dismisses typhlitis, or an inflammation of the cæcum proper, as "a doubtful and uncertain malady, the pathology of which is unknown, but which clinically is still recognised by authorities: a majority of the cases are unquestionably due to appendix disease."

EXAMINATION OF THE CÆCAL THEORY

When one considers the great frequency of this iliac phlegmon, or local peritonitis in the right iliac region, it is certainly remarkable that there should be so much doubt as to its pathology. The mystery that has so long surrounded it is to be explained by the extremely small mortality with which it is attended, and it follows from this small mortality that an absolute disproof of the alleged cæcal origin of some cases is not possible. This condition of perityphlitis stands apart from most other diseases having a like frequency of occurrence, in that its nature and origin must be inferred from the scanty premises afforded by rare and accidental opportunities for post-mortem observation and by the results of operative treatment. And it is clear that if an account of its pathology is to be approximately correct, we must supplement direct ocular evidence by the results of clinical work, checking the one by the other, and that neither of these fields of view will by itself prove sufficient.

Ulcerative perforation of the cæcum. That the cæcum has any connection with the severe degrees of peritonitis arising in its neighbourhood is now so generally denied that disproof is no longer necessary. The whole weight of clinical and pathological evidence is opposed to any such connection. It may almost be said with truth that every case of inflammatory disease in the right iliac fossa which has proved fatal has been found to be due to disease of the appendix. Osler has indeed met with two cases of perforation of a cæcal ulcer and pericæcal abscess. After extensive research into the literature of the subject Fitz could only find three cases, one due to a fishbone, another to a pin, the third from strangulation of the bowel. In fact, it will be generally allowed that a perforating ulceration of the cæcum, though it does certainly occur, is of so rare occurrence that it may be disregarded.

Non-perforating ulceration of the cæcum.

But the theory of a cæcal origin relates mainly to the milder forms of the disease, to which the name "stercoral typhlitis" was in former times habitually applied. It is not uncommon even at the present time to meet with an expression of this view. It is maintained that these mild cases depend on a non-perforating ulceration of the cæcum due to the retention of fæces—in fact, on the old-fashioned "stercoral ulcer." Is there any reason for continuing to believe in the existence of such an ulcer? Doubtless such an ulcer is a possibility, but it must be exceedingly rare, if we exclude those cases of ulceration in a distended cæcum which are due to continued obstruction at a point lower down in the bowel. Post-mortem records maintain an absolute silence about it. The very fact that perforation of a cæcal ulcer occurs with such infinite rarity serves to throw doubt on its existence.

The cæcum and appendix are almost identical in structure: there are differences only in the amount and arrangement of lymphatic tissue and muscle. Ulcers in the appendix due to fæcal impaction are, as will be shown later, of very common occurrence, and they perforate its walls with great frequency. It is a hard matter, then, to believe in a cæcal ulcer of similar origin which in only two or three recorded instances has ever perforated the peritoneal coat.

Haslam,[23] in arguing in favour of a true typhlitis, or inflammation of the cæcum, is more precise than most writers as to its morbid anatomy. He says, "The mucous membrane of the cæcum may be inflamed in association with a general colitis, or more frequently from irritation due to retention and impaction of fæces; the cæcum may be the seat of ulceration either from faecal irritation, tubercle, dysentery, typhoid, or foreign body." But when the inflammation of the cæcum is part of a general colitis, and when it occurs in the course of tubercle, dysentery, or typhoid, the symptoms are then those of the general disease, and are not those of the local condition in the right iliac fossa which is here under consideration. These general causes must then be excluded, and we are left stranded again with the theory of fæcal irritation and ulceration, in favour of which no actually observed fact has ever been recorded.

Catarrhal inflammation of the cæcum.

If then the theory of a stercoral ulcer be untenable, it may still be said that these cases depend on a catarrh of the mucous membrane of the cæcum unaccompanied by ulceration. This explanation is certainly most in accordance with the clinical history, for these cases of stercoral cæcitis are never fatal, and I believe are never supposed to end in suppuration. It is an explanation, however, which has no foundation on ocular evidence. It was inferred entirely from clinical observation, and the inference was made at a time when the importance of the appendix as a source of disease was not yet recognised. The theory was born of clinicians, yet the clinical evidence against it seems to me to be overwhelming. This condition is described as being manifested by pain which is trifling for a long time, by slight sensitiveness, by the absence or late occurrence of fever, and by the presence of a distinct, nodular, or doughy mass in the lumbar region. Osler describes it in much the same terms, but with a considerable shade of doubt. Haslam (*loc. cit.*) draws up a list of points which may serve to distinguish typhlitis from appendicitis.

Now it must be confessed that these symptoms, apart from the presence of a fæcal tumour, are in fact not to be distinguished from those of a mild degree of peritonitis arising round a diseased appendix. Just such a condition as this may often be seen to alternate with severe attacks of true appendicular peritonitis, as verified later by post-mortem examination. Even if we grant that the mass felt in the lumbar region is composed of fæces, still we know that in cases of true appendicular peritonitis a part of the hard iliac tumour is often composed of fæcal matter, which is retained in the cæcum through paralytic interference with its peristalsis. The presence of the fæcal matter is of secondary occurrence and of secondary importance. Hard fæcal lumps can often be felt in the cæcum, as also in the sigmoid flexure, in the course of hospital practice, but the patient has no special symptoms and knows nothing of their presence.

The position then is this. We know that a patient with inflammation of the appendix and slight local peritonitis around it may complain of but slight pain, and present but little tenderness and little or no fever, and may at the same time have a considerable area of hardness in the iliac region, which is doubtless in part due to a secondary paralytic retention of fæces. Only the strongest ocular evidence would justify us in reversing the explanation, and in explaining the condition as being due to a primary retention of fæces resulting in a secondary inflammation of the cæcum. And no such evidence is forthcoming.

On pathological grounds, then, it appears that there is no evidence in support of a cæcal origin to any of the inflammatory conditions which so frequently arise in the right iliac fossa. On the other hand, all clinical and pathological observations point to a primary affection of the appendix as underlying all degrees of this affection.

And, further, it may be taken as an established truth that the retrocæcal connective tissue (except in certain cases where the fascia covering the iliacus muscle is secondarily perforated) takes no part whatever in the inflammation, and that the inflammation is from first to last of the peritoneum alone.

Conclusions.

On reviewing the history of 264 cases the following conclusions seem inevitable:—

1. That there is a perfect gradation from the sudden general peritonitis, which is almost invariably fatal, down to the condition of slight pain and tenderness in the right iliac fossa, which may or may not keep a man from his day's work. That these several degrees alternate in the same patient, so that he may have one or more slight attacks and then succumb to a severe appendicular peritonitis, or a severe attack may be followed by several of the milder variety.

2. That there is no break in this chain, and no point at which one may say that appendicular disease ends and cæcal disease begins.

3. That, since whenever death occurred from any cause (37 instances in these 264 cases), the primary disease was in every case found to be in the appendix and not in the cæcum, the evidence in favour of an appendicular origin in all cases is overwhelming.

CHAPTER III

Comparative Anatomy of Cæcum and Appendix.
Anatomy of Appendix.—Length, size, lumen, position.
Arrangement of Peritoneum.—Cæcal fossæ, meso-appendix, cæcal peritoneum.
Structure of Appendix.

COMPARATIVE ANATOMY

THE key to the understanding of disease of the appendix lies in its developmental history. All the evidence which is to hand indicates that it is a rudimentary organ, a relic of a previous condition.

It is obsolete, functionless, and not in relation with the rest of the bodily economy, and in this way we may from general principles explain the surprising want of resistance to deleterious agents which a study of its diseases renders so prominent. To put it briefly, the comparative anatomy of the cæcum makes it clear that owing to certain altered conditions of life, mainly in the matter of food, man in his present condition finds himself provided with an ancestral cæcum which is too large for his wants. And the part of the cæcum which is discarded as unnecessary is that part of it which we call the vermiform appendix. Many reptiles have cæca, especially the Saurii. In birds the cæcum is present in all but a few families: it is generally paired, and may form a short appendage or a long tube. The Mammalia are well provided with cæca, and these are generally single. Its size in them may be shown to be dependent on the nature of the food. In the Carnivora it is short and sometimes completely absent (*ursina, mustelina*); it is very large in the Herbivora (Gegenbaur). The terminal portion is sometimes smaller than the base of the organ, as in various prosimiæ and many rodents; in the rabbit and hare this distal part is thickly studded with lymphoid follicles, and it may have an active part to play. But in various primates and in man the terminal part ceases to develop at an early period of embryonic life, and as the cæcum proper continues to grow, this part becomes more and more distinct as the vermiform appendix.

The comparative history of this part of the intestine is, in fact, repeated in the development of the individual. In the human embryo the cæcum gradually grows out from the primitive intestinal canal, forming at first a tube of uniform calibre. Subsequently the terminal part of the diverticulum ceases to grow in the same proportion as the rest, and remains undeveloped in the form of an appendix.

With this non-development there is bound up an inherent want of resistance to certain disease-producing agents.

ANATOMY OF APPENDIX

In the last ten years this functionless relic, the appendix, has received as much minute attention as many an organ of vital importance. Though no kind of knowledge may ever with truth be called superfluous, yet the cloud of anatomical details that have accumulated round it is such that a clear sight of the main points is somewhat obscured. Moreover, its gross anatomy has received such close scrutiny that a practical finality of description has been reached. Only such facts therefore will be detailed here as may be reasonably utilised in throwing light on the causation of the disease or in deciding on its proper treatment. As to the following observations, which are gleaned from various sources, I am convinced of their general truthfulness by repeated examinations, and on two points only will it be necessary to obtrude a personal experience.

Its length.

The length of the appendix is very variable. Bryant's[15] measurements establish the fact that the average length in the male is 3½ inches, in the female 3⅒ inches. This agrees with Ribbert's[16] statement. The extremes seem to be about 1 inch and 9 inches.

Ribbert gives a table showing that the length varies at different periods of life, the greatest length being found in early adult life, after which it tends to become shorter :—

At birth	average	2⅖	ctm.
Up to 5	,,	7⅔	,,
5 to 10	,,	9	,,
10 to 20	,,	9¾	,,
20 to 30	,,	9⅓	,,
30 to 40	,,	8¾	,,
40 to 60	,,	8⅓	,,
Over 60	,,	8¼	,,

He finds further that the relative length is much greater in infants than in adults; in the new-born its relation to the large intestine in length is as 1 to 10, in the adult as 1 to 20.

Its size. Bryant has shown that the diameter in the male averages $\frac{1}{10}$th of an inch more than in the female. The average diameter in the male appears to be $\frac{1}{4}$ of an inch.

A very important point was noted by him, that the greater the diameter of the appendix within apparently normal limits, the greater is the probability of the presence in it of fæcal or other matters. Thus when the appendix is $\frac{5}{16}$ths of an inch in diameter, 89 per cent. contain fæcal or other material; when $\frac{4}{16}$ths in diameter, 70 per cent.; when $\frac{3}{16}$ths in diameter, 60 per cent. And further he made the observation, which was to be expected from the foregoing data, that in males 70 per cent. of appendices contain fæcal matter with or without mucus, in females 35 per cent.

Condition of its lumen. In 100 bodies that I examined in the post-mortem room of St. Thomas's Hospital, the lumen was found to be obliterated locally in 4 and completely in 1. In the same number of bodies Treves [17] found local obliteration in 2 and complete obliteration in 1. Kelynack [18] found local obliteration in 21 per cent. and complete obliteration in 2 per cent. Rolleston (quoted by Kelynack) found local obliteration in 17 and complete obliteration in 4 out of 213 bodies. Ribbert (*loc. cit.*) states that he found 99 "strictures" in 400 bodies (25 per cent.), and he found further that their frequency of occurrence varies with the age in a very remarkable way.

At 1 to 10	years in	4	per cent. of bodies
,, 10 to 20	,,	11	,, ,,
,, 20 to 30	,,	17	,, ,,
,, 30 to 40	,,	25	,, ,,
,, 40 to 50	,,	27	,, ,,
,, 50 to 60	,,	36	,, ,,
,, 60 to 70	,,	53	,, ,,
,, 70 to 80	,,	58	,, ,,

The origin of these strictures will be discussed later.

Its position. A knowledge of the common positions assumed by the appendix is of use, partly because it throws some light on the variations seen in the results of its disease, but mainly because it may afford some guidance to an operating surgeon in his endeavour to pick out the appendix from a mass of adhesions and thickened omentum. Many tables have been compiled, some of them being exceedingly elaborate and minute, notably those of Bryant (*loc. cit.*) and of Turner.[19] It is possible that a more useful table would be one of a simpler and more practical character.

Treves (*loc. cit.*) found that "in the majority of cases in the adult body the appendix when examined *in situ* will be seen to lie behind the end of the ileum and its mesentery and to point in the direction of the spleen; in the only other position that may be said to be at all common (18 per cent.) the little tube ascends vertically behind the cæcum."

This is essentially in agreement with my own observation on 100 bodies of patients dead from other than appendicular disease :—

(1) Running up along the left border of the cæcum, or lying on the psoas pointing in an upward and inward direction (in six cases running at first downwards for a short distance before turning up on the psoas)		38
(2) Behind the cæcum :—		
(a) Curled up in this position	20	
(b) Running up straight for a variable distance (in one case the tip reaching the right kidney) ...		6—26
(3) Hanging down into the pelvis		17
(4) Running up on the outer side of the cæcum		9
(5) Running almost transversely inwards across the psoas ...		6
(6) Curled up in ileo-cæcal fossa or on the tip of the cæcum ...		4

Thus it may be said roughly that in 38 per cent. it runs upwards and inwards, in 26 per cent. it lies behind the cæcum, and in 17 per cent. it dips into the pelvis. At the same time it must be remembered that it may be placed anywhere, and it is hardly necessary for practical purposes to enumerate all recorded instances of unusual situations, beyond pointing out that the tube may have such an unusual length that it has been found lying with its tip upon the left psoas (Treves). Fowler[20] records three cases where the appendix lay on the left of the middle line of the body. Its tip also sometimes contracts adhesions to rectum, bladder, broad ligament, colon, or mesentery, so that a loop is formed, with the possibility of subsequent strangulation of the bowel.

The safest plan in searching for the appendix is to follow downwards the anterior muscular band of the cæcum.

It remains to mention that Bryant's table shows that there is no substantial difference in the position of the appendix in the two sexes.

Peritoneum around cæcum and appendix.

Cæcal fossæ.

The arrangement of the peritoneum around the cæcum must be understood in order to comprehend fully the various hiding-places of the appendix. Without going at any length into this subject, an admirable paper by Lockwood and Rolleston[21] may be referred to as giving the fullest information on the matter. They describe three fossæ in this region, the two latter of which are certainly of importance as regards the subject of this paper.

(1) *Ileo-colic fossa* is the angle formed by the ileum with the colon. It has the ascending colon for its outer boundary, the ileum for its lower boundary, and the mesentery for its

floor. Very often a fold of peritoneum, carrying the anterior ileo-cæcal artery in it, roofs in the angle, which thus becomes a veritable pocket with its mouth looking upward and to the left. It is not probable that this fossa can have any importance in relation to disease of the appendix.

(2) *Ileo-cæcal fossa* is situated behind the angle of junction of the ileum and cæcum, which have both to be lifted up in order to see its mouth. It is bounded on the right by the mesentery of the ascending colon, and on the left by the mesentery. It may reach halfway up the ascending colon, and end close to the kidney and duodenum. This fossa is often divided by the mesentery of the appendix into two compartments one above the other, the superior and inferior ileo-cæcal fossæ. The superior of these two fossæ again may be roofed in by the ileo-cæcal fold. This ileo-cæcal fossa is by no means an uncommon situation of the appendix, and its existence probably explains certain differences in the results of disease which will be subsequently alluded to.

(3) *Subcæcal fossa* is situated immediately under the cæcum, which has to be lifted up to bring it into view. It is less often present than either of the other two fossæ. It separates the layers of the meso-colon, and its mouth is at the junction of the cæcum and colon.

This fossa would be of more clinical importance if a meso-cæcum were at all common. Its mouth would then lie flush with the tip of the cæcum close to the origin of the appendix, and this condition Lockwood and Rolleston describe, but I have never myself been able to find a cæcum which was not completely invested by peritoneum.

The meso-appendix.

The meso-appendix is described by Treves as being a fairly constant peritoneal fold. It comes off from the left or under layer of the mesentery of the ileum, and if put on the stretch it seems to come off at right angles to the mesentery, and to lie in the plane of the iliac fossa. It is triangular in shape. To the right the meso-appendix runs up to the ileo-cæcal junction, on the left it ends in a sharp free edge which contains the main artery of the appendix, a branch of the ileo colic. It is however very variable in extent. (1) It is sometimes absent, so that the appendix is quite free in the abdomen. (2) In the fœtus it may extend to the tip of appendix (Treves), and Lockwood and Rolleston found one specimen in which it reached to the free end of an appendix four inches long. (3) As a rule it has the appearance of being too short for the appendix, and this tube is thus commonly curled spirally and sometimes bent upon itself, and in these cases it cannot be straightened until the meso-appendix is divided. Bryant (*loc. cit.*) found that in 40 per cent. of bodies one half or more of the length of the tube was completely surrounded by peritoneum and had no mesentery. (4) Again, Lockwood and Rolleston have shown how in the developmental descent of the cæcum into the iliac fossa the meso-appendix may become obliterated by its peritoneum having been spread out in the iliac fossa by the traction of the gubernaculum. The base of the appendix may thus come to be bound as it were firmly down to the peritoneum lining the iliac fossa. (5) Finally, Treves (*loc. cit.*) has shown how the cæcum as it grows tends to encroach upon the peritoneum of the neighbouring part, clothing itself as it were with part of the mesentery of the ileum and of the mesentery of the appendix. All stages of this encroachment are depicted in his Hunterian Lectures: in the last stage the appendix has scarcely any mesentery of its own, but is adherent in a vertical line to the posterior wall of the cæcum, the tip however as a rule remaining free. These last two variations in the meso-appendix are of great clinical importance, and they also serve to make us extremely cautious in fully accepting the statements of those observers, who in such a large percentage of bodies find evidence of supposed previous peritonitis in local adhesions around the appendix.

It follows from what has been said that, though the appendix may in part be closely bound to the peritoneum lining the iliac fossa or may in part be closely adherent to the posterior surface of the cæcum, yet in any other sense an "extra-peritoneal appendix" is an anatomical impossibility, and it is difficult to understand the frequency with which this expression is met with.

Peritoneum of cæcum.

The frequency of a meso-cæcum was first called in question by Treves, who in 100 bodies never found a cæcum which was not entirely invested by peritoneum. He was disposed to doubt the existence of such a fold. Lockwood and Rolleston make use of the term meso-cæcum without describing it specifically. I have never been able to find one in the post-mortem room. In fact, the mobility of the cæcum and its variability in position are points with which in regular post-mortem work one is constantly impressed.

Position of cæcum

The cæcum as a rule lies on the psoas with its apex projecting just beyond its inner edge, corresponding to a point slightly to the inner side of the middle of Poupart's ligament. But in the fœtus it has a higher and more central position, and this fœtal condition may be persistent. Thus in three cases recorded by Fowler (*loc. cit.*) it lay under the umbilicus. It is not very uncommon in the post-mortem room to find it lying in the right loin, having no relation with the iliac fossa; and it may be found lying on the right kidney. Lenander found it lying against the spleen in a boy aged sixteen.

These variations in its position are important, since in such cases the appendix must be similarly misplaced.

STRUCTURE OF APPENDIX.

The structure of the appendix on the whole differs but little from that of the cæcum.

Mucosa. The general substance of the mucous membrane is composed of a delicate retiform tissue. This holds in its meshes a varying number of lymphoid cells, which may here and there be aggregated into small dense masses. Running up in this retiform tissue towards the surface may be seen numerous parallel rows of elongated nuclei, representing capillaries and lymphatics.

This retiform tissue is bounded towards the lumen of the tube by a definite basement-membrane, which can in nearly all cases be clearly distinguished. On this basement-membrane is seated a columnar epithelium, many of the cells of which show the common goblet-form.

Dipping down into the retiform tissue are numerous tubular glands, the crypts of Lieberkühn, lined by a columnar epithelium and a basement-membrane continuous with these structures on the general surface. These crypts vary somewhat in number, but as a rule they are so closely placed as to be separated only by narrow columns of the general retiform tissue of the mucosa. Rarely they divide into two at their lower ends.

As a rule the mucosa is bounded on its deep aspect by a more or less continuous narrow circular strand of muscle, the "muscularis mucosæ." And the crypts of Lieberkühn commonly dip down to the immediate neighbourhood of this layer.

In many cases, as has been mentioned, there are small aggregations of lymphatic tissue in the inter-glandular columns of retiform tissue. In every case examined there are also large aggregations of lymphatic tissue (lymphoid follicles) which may lie in the deepest part of the mucosa, but as a rule lie in the submucosa, being in these cases separated off from the mucosa proper by the muscularis mucosæ. Sometimes a few strands of tissue similar to the muscularis mucosæ can be seen to lie between the lymphoid follicles and the submucosa. The supporting tissue of these follicles is continuous with the retiform tissue of the mucosa. They are formed of dense clumps of lymphoid cells. These clumps vary greatly in number. Sometimes two or three only are seen in a transverse section of an appendix, sometimes eight or ten, rarely they are so numerous as to form a complete ring round the tube. The specimens examined do not indicate that the amount of lymphatic tissue has any relation to the age of the individual, but only the examination of a very large number of normal cases would justify any definite statement on this point.

The free surface of the epithelial lining is found to be constantly beset by microbes in great number, both cocci and rods of various sizes.

As seen *in situ* with the naked eye, the mucosa is a soft thick grey pulpy membrane, in most cases showing numerous dark points which are the lymphatic follicles.

There is as a rule a slight duplicature of the mucous membrane at the orifice of the tube, seen as a low edge sometimes running halfway round the lumen. It was described originally by Gerlach. It is in no sense a valve, but it tends to make more difficult the communication between cæcum and appendix in both directions.

Submucosa. Under the mucous membrane, and often embedding the lymphoid follicles, lies the submucous coat. This consists of loose wavy strands of areolar tissue. In it are seen numerous small arteries and veins dividing for the supply of the mucosa, and it generally contains a certain amount of fat.

Muscular coats. The inner muscular coat consists of plain muscular fibres disposed in a circular manner. This is a stout layer, though variable in thickness. Even in perfectly normal specimens it may form a third of the thickness of the tube. It is difficult to understand the statement of Austin Flint[22] that circular muscular fibres do not exist.

The outer muscular coat is generally rather thinner than the inner coat. It consists of plain muscular fibres arranged in the long axis of the tube. As a rule it forms a continuous coat, but sometimes it shows a tendency to be aggregated at certain spots, as is the case in the cæcum.

Peritoneum. The peritoneal coat of the appendix needs no special description. It consists of a groundwork of connective tissue covered with a layer of flattened cells.

CHAPTER IV

Frequency of Disease of the Appendix.
Classification of Diseases of the Appendix.
Catarrhal Appendicitis.—Acute, chronic ; obliteration, stricture, cystic distension by stricture and kinking.
Ulcerative Appendicitis.—Fæcal concretion, foreign body.
Infective Appendicitis.—Naked-eye appearance, microscopical appearance ; necrosis of and suppuration in the wall of the appendix ; bacterial origin ; bacteriology of the appendix ; invasion of intestinal wall by bacteria ; special liability of the appendix to such invasion ; conclusions.
Other Diseases of the Appendix.

THE PART PLAYED BY THE APPENDIX

If the cæcum is to be entirely eliminated, and if this local form of peritonitis which we call perityphlitis is in all cases to be ascribed to the appendix, it is a necessary corollary (considering the frequency of its occurrence) that in the general post-mortem work of a large hospital there should be found abundant evidence of appendicular disease. And this is found to be the case. Tungel, quoted by Fitz, during a period of two years at the Hamburg Hospital, found thirty instances of partial or complete obliteration of the appendix, forty-three cases of catarrh and fæcal concretions, twelve of abnormal adhesions, and eleven of tubercular ulcers. Toft (*cf.* the paper by With, *loc. cit.*) found the appendix diseased in 100 out of 300 post-mortem examinations.

Now it is not certain whether these figures are founded on naked-eye or microscopical examination, and their value consequently is doubtful. I have therefore examined the appendix at this hospital in 100 bodies of all ages taken at random, where death occurred from some other cause than disease of this organ, and if there was any naked-eye appearance of disease the suspected organ was submitted to microscopical examination. (In all cases the specimens were embedded in celloidin.) In these 100 cases there were sixteen in which there was past or present disease of the appendix to be seen. (There were also two cases of tubercular and one of dysenteric ulcer, which may be disregarded as having no connection with the present subject.) In seven of these sixteen cases there was chronic catarrh, with commencing obliteration of the lumen in one of them: in four the lumen was wholly or in part obliterated by fibrous adhesion of its walls; in five there was ulceration due to the pressure of a fæcal mass. In two of the cases of catarrh the appendix was bound down by old peritonitic adhesions, and this condition was present in all four of the obliteration cases.

It is not safe to draw any dogmatic conclusion from 100 cases, but so far as it goes there is here (16 per cent.) ample evidence that appendicular disease is at any rate of frequent occurrence; and this frequency, moreover, is numerically sufficient to justify us in regarding the appendix as the sole cause of all cases of perityphlitis, mild or severe.

Classification of the diseases of the appendix.

Sufficient data have now been collected to warrant an attempt to frame a comprehensive account of the inflammatory affections of the appendix. These may be realised to a great extent from microscopical examination, and the material that is here utilised is drawn from operations and post-mortem examinations at St. Thomas's Hospital.

Our knowledge of them, however, does not admit of a scientific classification. The old division of appendicitis into a "perforative" and a "non-perforative" form has lost its value for clinical use, now that we know that both an acute general peritonitis and a perityphlitic abscess may result from non-perforative disease of the appendix. But examination of a large number of specimens makes it clear that Osler's division of appendicitis into a "catarrhal" and an "ulcerative form" is a good one. Though these two do to a certain extent overlap, inasmuch as catarrh may end in ulceration, and both conditions may exist side by side in the same specimen, yet is this distinction nevertheless useful, since they arise from different causes. The cause of the "catarrhal" form we do not exactly know, but it is clear that it is not dependent on the presence of fæcal or other extraneous matter; while the "ulcerative" form, on the other hand, is produced mainly by the pressure of a fæcal concretion or a foreign body. Equally from both these forms may arise every grade of peritonitis.

In a large number of cases it happens that the peritonitis arises through a local or general "necrosis" of the appendix, which may be the primary condition or may arise as a result of previous catarrh or ulceration. There is reason to believe that in these cases the necrosis is dependent upon a bacterial invasion of the tissues of the appendix-wall and consequently a third form, viz. "infective appendicitis," may well be considered under a separate head.

(A) CATARRHAL APPENDICITIS

A catarrhal inflammation of the appendix is probably of very frequent occurrence, but its exact frequency cannot be determined, for in itself it produces no symptoms, and doubtless, like catarrhs of other mucous membranes, it may subside without leaving any trace.

A chronic catarrh is, however, very commonly found in general post-mortem work, and it is easily detected, for it gives rise to a recognisable thickening of the whole wall. In this condition the appendix is stiff, and on section tends to maintain its cylindrical shape.

Simple catarrh of the appendix is independent of the presence of fæcal matter. In some of the specimens examined there was a little soft pasty fæcal matter here and there mingled with mucus, but in most of them the lumen was quite clear, and it is not to be supposed that the mere contact of fæces with the mucous membrane has any deleterious effect.

Acute catarrhal appendicitis.

The actual changes are those of inflammation of mucous membranes elsewhere. There occurs a rapid shedding of the epithelium of the general surface with detachment and extrusion of the epithelial lining of the crypts of Lieberkühn. There is some leucocytal infiltration of the delicate retiform tissue which forms the groundwork of the mucous membrane, and by the swelling thus produced there is pressure upon and even obliteration of many of the crypts. The lumen is found to contain abundance of leucocytes, granular débris, mucus, and casts of the interior of the crypts, and the whole of this material is sometimes moulded into a definite central mass by the muscular movements of the tube.

This stage is shown in Fig. 1.

As the process continues, the basement-membrane is destroyed, and the leucocytal infiltration of the retiform tissue becomes more marked. So that the mucosa comes to consist of a dense cellular layer, having a ragged raw internal surface, and containing pockets in which lie the degenerating remnants of epithelial cells. This is shown in Fig. 2.

Perfect recovery by new growth of epithelium is at first possible, but the shedding may be so complete over so large an area that the denuded surface cannot be covered anew. The inner part of the mucosa now comes to consist of granulation-tissue, in which the remains of a few of the once plentiful crypts may be distinguished, as in Fig. 3.

This condition is probably, as will be shown later, a fertile source of local peritonitis, but it is fortunately in many cases the precursor of complete immunity from such attacks. If by the pressure of the surrounding parts the opposed granulating

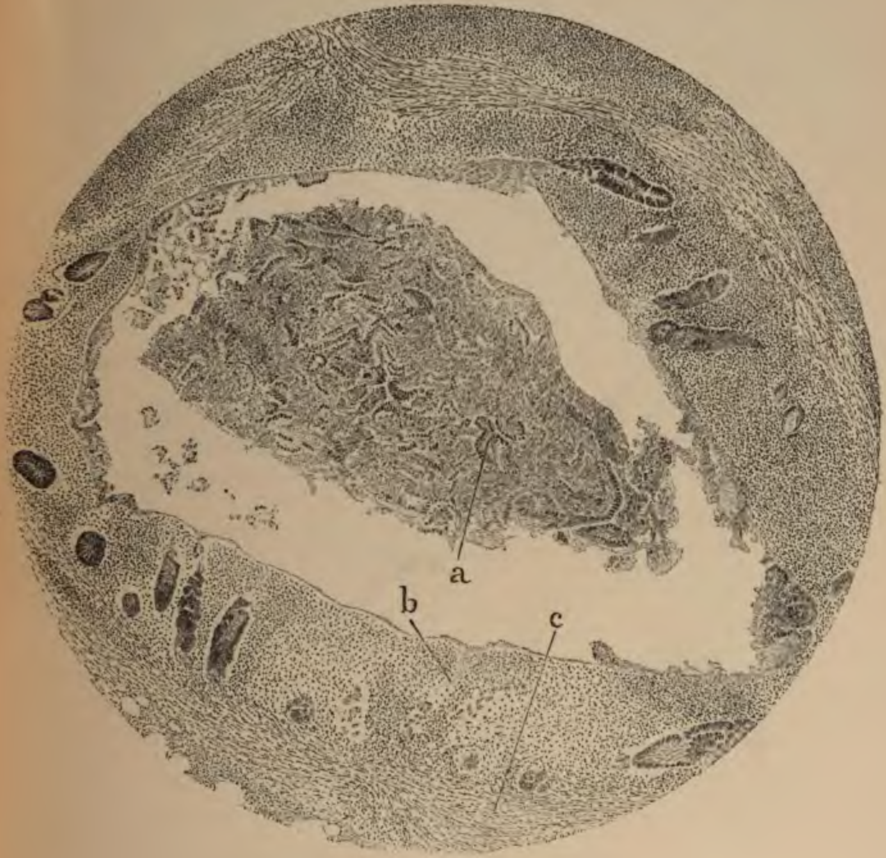

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FIG. 1.—CATARRHAL APPENDICITIS.

Transverse section of an appendix which was bound down by adhesions along the inner border of the cæcum. Part only of the thickness of the wall is here shown.

The general epithelial lining has been almost entirely shed, only a few degenerated cells and granular débris remaining. The basement-membrane is for the most part intact.

The central mass lying in the lumen is composed of granular material, leucocytes, and epithelial cells, with a few dark granules of fæcal matter. In it are many complete epithelial casts of the tubular crypts of Lieberkühn.

The crypts have been largely obliterated, and the epithelium of the surviving crypts is degenerated.

a. Epithelial cast embedded in the central mass.
b. Empty socket of a crypt.
c. Submucous areolar tissue.

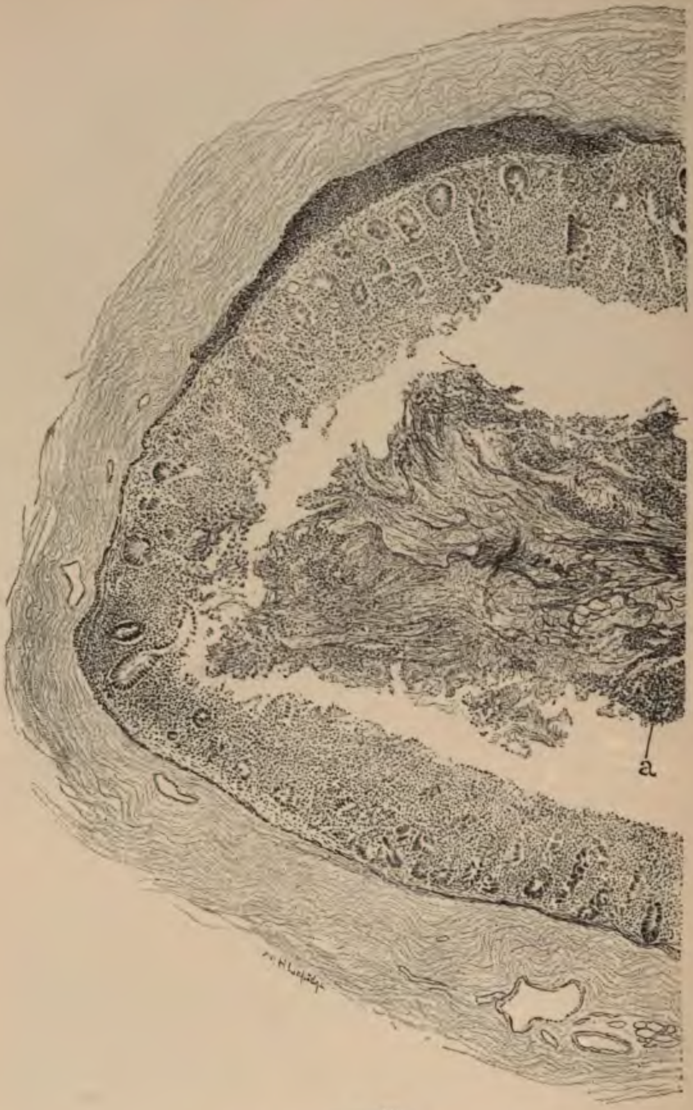

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FIG. 2.—CATARRHAL APPENDICITIS.

Transverse section of an appendix, showing only the mucosa and submucosa.

The epithelial elements of the mucosa are degenerated and largely shed. The basement-membrane has been destroyed.

a. Mass of cellular elements embedded in a large central collection of mucus.

surfaces are brought into contact, and if the whole organ remains at rest, union may take place, and the appendix as a source of disease then ceases to exist. The bearing of this on treatment will be referred to subsequently. The commencement of this process of union and obliteration is seen in Fig. 4, and also in Fig. 22.

As a final result the lumen may come to be occupied entirely by fibrous tissue springing directly from the submucous areolar tissue, and natural cure is then complete. An appendix obliterated in this way is shown in Fig. 5.

Chronic appendicitis.

In many cases, however, the condition lapses into one of chronic appendicitis, and obliteration of the lumen is delayed or rendered impossible by reason of the great thickening of the wall of the tube, which resists apposition of the granulating surfaces. While the inner surface continues as a pus-secreting granulation-tissue, the muscular coats may often be seen to be infiltrated with leucocytes and connective-tissue cells, and in some cases there is great increase of the fibrous element of these coats, insomuch that the tube becomes rigid and incollapsible. The wall of one specimen was an eighth of an inch in thickness, being about double that of the healthy tube, and in another case the thickness of the muscular coat alone amounted to one tenth of an inch. In this incollapsibility probably lies the clue to some cases of "relapsing perityphlitis," of which instances will be subsequently given.

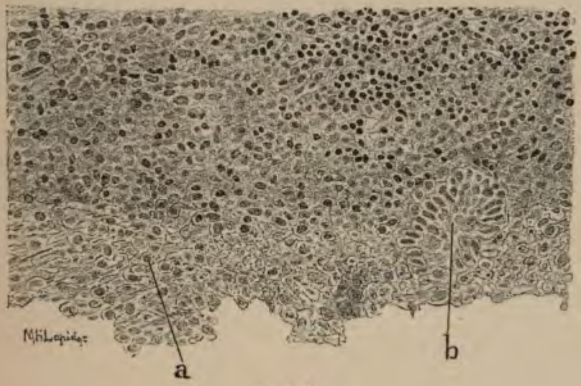

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FIG. 3.—CHRONIC APPENDICITIS.

Transverse section of the inner part of the mucous membrane of an appendix, which was embedded in peritonitic adhesions.

a. Granulation tissue.

b. Remains of a crypt.

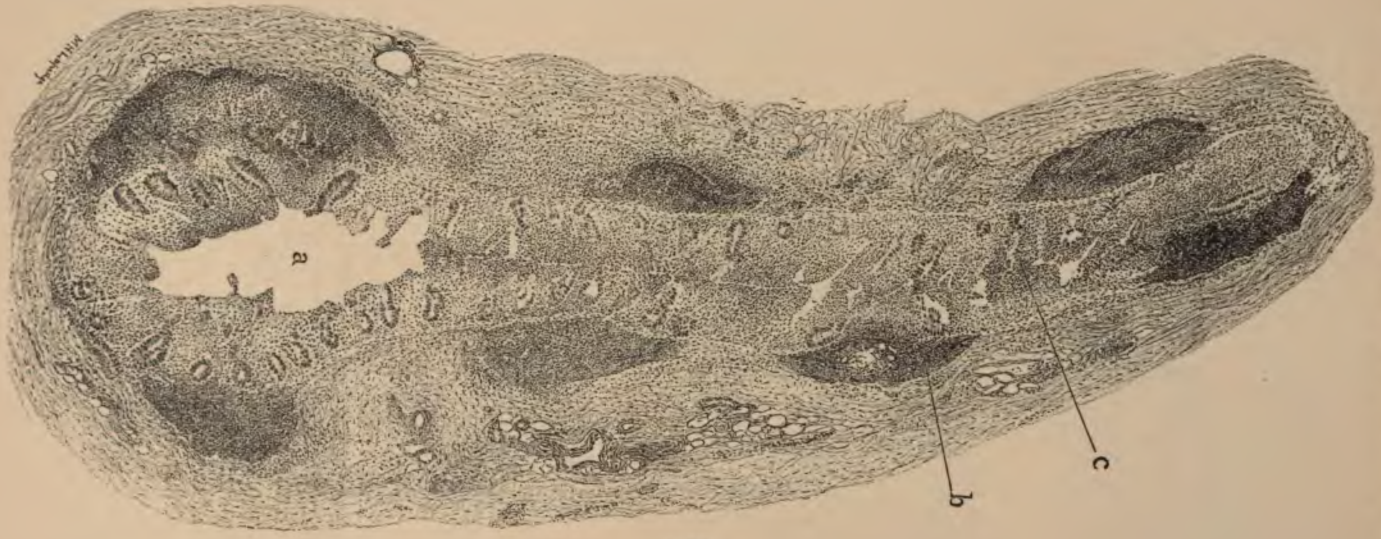

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FIG. 4.—COMMENCING OBLITERATION OF APPENDIX.

Transverse section of an appendix near the base, showing the commencement of union between opposed granulating surfaces. Only part of the thickness of the wall is shown.

The general epithelial lining and the basement-membrane have been destroyed. The epithelium of the crypts has been largely shed, especially at the upper end of the figure, where union is most advanced.

a. Remains of the lumen of the tube. *b*. One of seven nodules of lymphoid tissue. *c*. Point where union is complete.

Stricture and cystic distension of the appendix.

Obliteration of the lumen of the appendix does not however in all cases extend throughout the whole length of the tube. It is not uncommon to find a partial obliteration only, so that a local impermeable stricture may result. This stricture may be at any spot, but it is generally near the cæcal end, probably because the degree of rest which the organ may attain (and

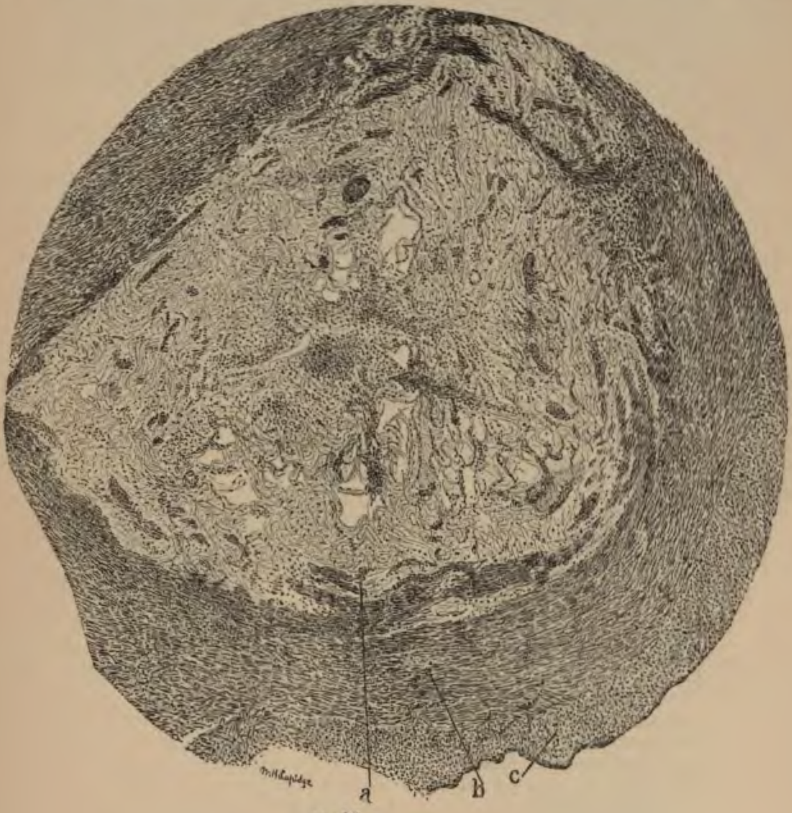

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Fig. 5.—Complete Obliteration of Appendix.

Transverse section of an appendix which was firmly adherent to the psoas and to the omentum. The appendix was a solid cord from end to end, without any trace of epithelium or mucous membrane.

The whole thickness of the wall is shown only in the lower right quadrant of the figure.

The lumen is occupied by fibrous tissue.

a. Remains of submucous areolar tissue.
b. Internal muscular coat.
c. External muscular coat.

consequently the possibility of cohesion of its wall) increases from the tip towards the attached end. In some cases two or more such strictures may be found at different points.

Complete obliteration of the whole length of the lumen can hardly arise in any other way than by the shedding of the epithelium and by the conversion of the mucosa into granulation-tissue, as the result of a chronic inflammation. And I think there can be little doubt but that the majority of localised strictures have a similar origin.

The frequency with which these strictures have been found has already been mentioned.

The part beyond the stricture may become distended by its own secretion into a veritable cyst, which sometimes assumes large dimensions.

It is possible that in the two following cases we may be justified in tracing the sequence of events, by which a cystic appendix is the outcome of a preceding catarrhal inflammation, though of course it is equally possible that in both cases all the attacks of peritonitis, early and late, were induced by a cystic distension already in existence.

CASE I.

Fatal general peritonitis due to perforation of a cystic appendix, preceded by an early slight attack of local peritonitis.

H. N., a clerk, aged 20, had a slight attack of perityphlitis in October 1887, marked by sudden onset of pain in the right iliac region with vomiting, and this kept him from work for a few days only. Nine months later he died from general suppurative peritonitis after four days' illness. His appendix was found to be 2½ inches long; one third of an inch at the cæcal end was a solid cord, and the terminal part, 2 inches long, was distended into an oval cyst three quarters of an inch in breadth. This cyst contained purulent fluid, its inner surface was uniformly ulcerated, and perforation had taken place in the floor of an ulcer close to the point of obliteration. It is represented in Fig. 6, which is partly diagrammatic, inasmuch as the perforation is omitted.

CASE II.

Fatal general peritonitis due to perforation of a cystic appendix preceded by three attacks of more localised peritonitis.

E. W., aged 28, had four severe attacks in eight months, during the first and last of which she was a patient in St. Thomas's Hospital. In the last attack she died. The appendix was found to be strictured at a point one eighth of an inch from the cæcum; the rest of it, two inches in length, had been obviously distended; it was thickened and black, and showed a ragged ulcerated hole about its middle, the whole of the inner surface having a worm-eaten appearance. One can only regret that this appendix was not excised after the first attack.

In Fig. 7 is shown an appendix taken from the body of a man who died from general appendicular peritonitis. (I am indebted to Mr. Ballance for the use of the drawing.) It shows two narrowed parts with two dilatations, perforation of one of which was the cause of death.

A good example has been published by Bland-Sutton[24]

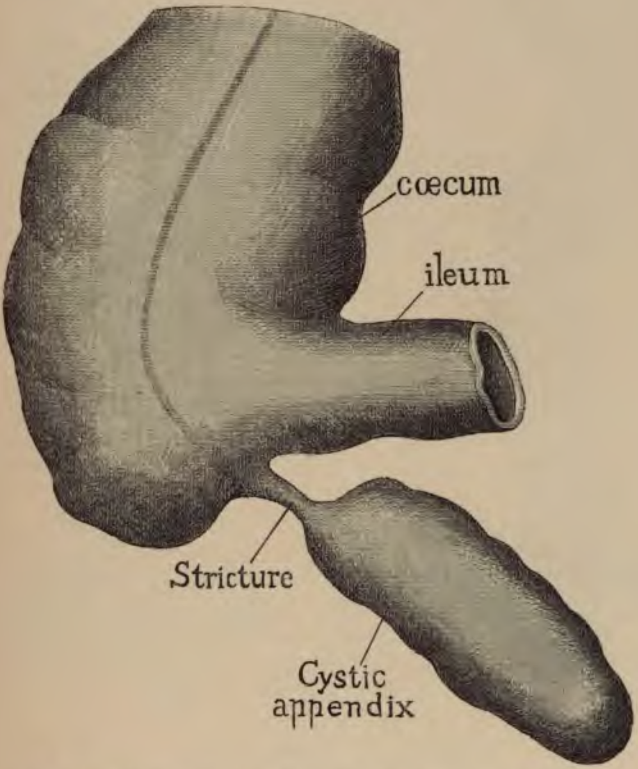

Fig. 6.—Stricture and Cystic Distension of Appendix.

The patient, aged twenty-two, had four attacks of perityphlitis in three years, in the last of which the appendix was excised.

"It was 6 cm. long; for 2 cm. at the cæcal end it was soft and pliable like a normal appendix, the rest was as round and firm as a fat earth-worm, and at the junction of soft and hard parts was an oval swelling. The cæcal end was found to be completely strictured, and the part beyond the stricture was filled with pus. The walls were slightly thickened, and the mucous membrane was completely destroyed."

CHAPTER IV

Frequency of Disease of the Appendix.
Classification of Diseases of the Appendix.
Catarrhal Appendicitis.—Acute, chronic ; obliteration, stricture, cystic distension by stricture and kinking.
Ulcerative Appendicitis.—Fæcal concretion, foreign body.
Infective Appendicitis.—Naked-eye appearance, microscopical appearance ; necrosis of and suppuration in the wall of the appendix ; bacterial origin ; bacteriology of the appendix ; invasion of intestinal wall by bacteria ; special liability of the appendix to such invasion ; conclusions.
Other Diseases of the Appendix.

THE PART PLAYED BY THE APPENDIX

IF the cæcum is to be entirely eliminated, and if this local form of peritonitis which we call perityphlitis is in all cases to be ascribed to the appendix, it is a necessary corollary (considering the frequency of its occurrence) that in the general post-mortem work of a large hospital there should be found abundant evidence of appendicular disease. And this is found to be the case. Tungel, quoted by Fitz, during a period of two years at the Hamburg Hospital, found thirty instances of partial or complete obliteration of the appendix, forty-three cases of catarrh and fæcal concretions, twelve of abnormal adhesions, and eleven of tubercular ulcers. Toft (*cf.* the paper by With, *loc. cit.*) found the appendix diseased in 100 out of 300 post-mortem examinations.

Now it is not certain whether these figures are founded on naked-eye or microscopical examination, and their value consequently is doubtful. I have therefore examined the appendix at this hospital in 100 bodies of all ages taken at random, where death occurred from some other cause than disease of this organ, and if there was any naked-eye appearance of disease the suspected organ was submitted to microscopical examination. (In all cases the specimens were embedded in celloidin.) In these 100 cases there were sixteen in which there was past or present disease of the appendix to be seen. (There were also two cases of tubercular and one of dysenteric ulcer, which may be disregarded as having no connection with the present subject.) In seven of these sixteen cases there was chronic catarrh, with commencing obliteration of the lumen in one of them: in four the lumen was wholly or in part obliterated by fibrous adhesion of its walls; in five there was ulceration due to the pressure of a fæcal mass. In two of the cases of catarrh the appendix was bound down by old peritonitic adhesions, and this condition was present in all four of the obliteration cases.

It is not safe to draw any dogmatic conclusion from 100 cases, but so far as it goes there is here (16 per cent.) ample evidence that appendicular disease is at any rate of frequent occurrence; and this frequency, moreover, is numerically sufficient to justify us in regarding the appendix as the sole cause of all cases of perityphlitis, mild or severe.

Classification of the diseases of the appendix.

Sufficient data have now been collected to warrant an attempt to frame a comprehensive account of the inflammatory affections of the appendix. These may be realised to a great extent from microscopical examination, and the material that is here utilised is drawn from operations and post-mortem examinations at St. Thomas's Hospital.

Our knowledge of them, however, does not admit of a scientific classification. The old division of appendicitis into a "perforative" and a "non-perforative" form has lost its value for clinical use, now that we know that both an acute general peritonitis and a perityphlitic abscess may result from non-perforative disease of the appendix. But examination of a large number of specimens makes it clear that Osler's division of appendicitis into a "catarrhal" and an "ulcerative form" is a good one. Though these two do to a certain extent overlap, inasmuch as catarrh may end in ulceration, and both conditions may exist side by side in the same specimen, yet is this distinction nevertheless useful, since they arise from different causes. The cause of the "catarrhal" form we do not exactly know, but it is clear that it is not dependent on the presence of fæcal or other extraneous matter; while the "ulcerative" form, on the other hand, is produced mainly by the pressure of a fæcal concretion or a foreign body. Equally from both these forms may arise every grade of peritonitis.

In a large number of cases it happens that the peritonitis arises through a local or general "necrosis" of the appendix, which may be the primary condition or may arise as a result of previous catarrh or ulceration. There is reason to believe that in these cases the necrosis is dependent upon a bacterial invasion of the tissues of the appendix-wall and consequently a third form, viz. "infective appendicitis," may well be considered under a separate head.

The relative frequency of the presence of a fæcal concretion and a foreign body was at one time grotesquely distorted. Foreign bodies are certainly found as the cause of an ulcerative perforating appendicitis, and they are sometimes found as the nucleus of a fæcal concretion; but in a vast majority of instances it is a mass of inspissated fæces which is the cause of the trouble.

The theory that the fæcal concretion which is so often found in the appendix has been formed in the cæcum, and has been subsequently forced into the appendix, has no foundation in fact. Although the appendix does not lie in the direct path by which the intestinal contents are forwarded, yet it is very rare to find one that does not contain sufficient fæcal matter to be recognised with the naked eye. No doubt every contraction of the cæcal wall upon its contents may tend to express a small quantity into the orifice of the appendix, which may be subsequently returned by the peristaltic contraction of that tube. Especially will this happen when the cæcal contractions are vigorous and the upward movement of the cæcal contents are more than commonly opposed by their bulk and consistence, as in the condition of chronic constipation.

The actual development of what we speak of as a fæcal concretion—that is, the acquisition by fæcal matter of a certain bulk and a certain degree of hardness—depends probably on the length of time during which such fæcal matter is allowed to lie unmoved in the appendix. The longer it lies undisturbed, the more water will it lose.

The mere presence of fæces in the appendix can be seen microscopically over and over again to have no injurious effect on the epithelium. Ulceration does not occur until the fæcal mass acquires a certain degree of hardness and comes to exercise a considerable degree of pressure upon the mucous membrane.

The hardness of the fæcal mass and its pressure against the wall of the appendix depend on the peristaltic contraction of the tube. The appendix is richly supplied with muscle, considering that it plays no part in forwarding the intestinal contents. It is not uncommon to find a mass of fæces which has been driven down into the blind end of the tube with such force that the tip assumes a bulbous appearance: on opening it and shelling out the mass, a fossa is seen, of which the floor may be found to be ulcerated. Two or three such masses may be found at different points, each one indicated by an annular dilatation of the unopened tube, which has thus a moniliform appearance. At each such point may be found ulceration. The barleycorn shape of these masses, with their thick substantial middle and tapering ends, indicates the action of waves of peristaltic contraction running up to the mass from both directions.

If the mass remains long in its bed, it cannot be removed, except by its breaking up, and possibly this happens. In many cases, however, a deposition of lime salts, mainly carbonate and phosphate of lime, takes place upon the surface of the concretion. It grows in size and hardness, and its destructive effect on the mucous membrane is thereby greatly intensified; but a fæcal mass may ulcerate through the entire wall and be extruded into the peritoneal cavity, while still purely fæcal and still in a plastic condition.

As the fæces begin to exercise pressure on the wall, the first effect is a disturbance of the lining epithelium. This is shown in Fig. 8, where three fæcal particles are seen to have been driven through the basement-membrane into the retiform tissue. The section is taken at the level of the tapering end of an oat-shaped concretion, which is omitted in the drawing.

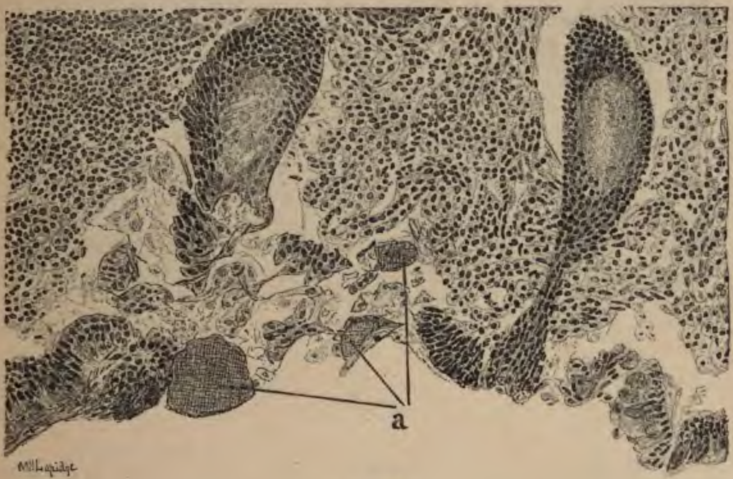

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FIG. 8.

Transverse section of the mucous membrane of an appendix just below a firmly fixed fæcal concretion.

The epithelial lining and the basement-membrane are healthy and intact, except in the centre, where three fæcal particles are seen to have been driven through them.

a. Fæcal particles.

As the process continues, the epithelium is completely destroyed; and the mucous membrane, the submucous tissue, and finally the muscular coats disappear before the advancing pressure. In Fig. 9, which shows the great pressure exercised by a large concretion (only part of which is here represented), the mucous membrane is much thinned, and to the right of the section it has been completely destroyed.

In some cases it is hardly correct to describe this process as one of ulceration. It appears to be rather an atrophy produced by the pressure, and evidence of inflammation may be scanty or absent.

Microscopical examination shows that the peritoneal coat of the appendix is far more tolerant of this distending process than might have been supposed. It is not uncommon to meet with an appendix distended by a fæcal concretion, so that its wall consists only of thinned-out muscular tissue, and yet the peritoneum may show no sign of past or present inflammation.

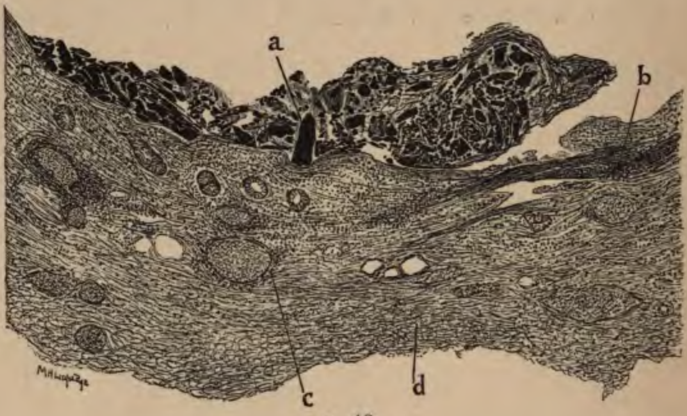

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FIG. 9.

Transverse section of part of the wall of the same appendix, taken at a point where the pressure of a fæcal concretion was very great. There was another similar concretion a little lower down in this case, and this had ruptured the coats, setting up a localised suppuration around it. The abscess was opened, the appendix removed, and the patient made a good recovery.

Only a small part of the concretion is shown here. The epithelial lining has been destroyed, and to the right of the figure the mucous membrane has completely disappeared, the concretion resting on the submucous tissue.

a. Indriven fæcal particle at the periphery of the concretion.
b. Condensed submucous areolar tissue.
c. Internal muscular coat.
d. External muscular coat.

A case was recorded by Blackadder[28] in 1824, where an earthy-looking concretion, the size of a thrush's egg, was found in the appendix of a man who died from phthisis, but he had no sign or symptom of any affection in the right iliac region.

I have not had an opportunity of examining microscopically the appendix in a case of disease resulting from the presence of a foreign body, but there is every reason to believe that the result is of the same character. There appears also to be the same tolerance of a foreign body as of a concretion. Thus in a case mentioned by Lewis (*loc. cit.*) 122 robin-shot were found in the appendix of a man aged 88 who had died from pneumothorax. It is stated that he was very fond of game. There had been no symptom during life, and there were no adhesions or other sign of disease about the appendix.

Fæcal concretions.

There is no doubt that fæcal masses, hard enough to have a deleterious effect on the mucous membrane, are of very frequent occurrence, but it must be remembered that most statistics on this head are taken from the results of operations on perityphlitic abscesses or from the bodies of those who have died of appendicular peritonitis.

Thus, of twenty-seven fatal cases of general appendicular peritonitis occurring in St. Thomas's Hospital, the cause was found to be a fæcal concretion in eleven, and in six out of thirty-three cases of local perityphlitic abscess a concretion was found in the pus.

Fitz (*loc. cit.*) found fæcal masses in 47 per cent. of 152 cases of perforative appendicitis. Matterstock[29] found concretions in 53 per cent. of his 169 cases. Krafft[30] found them in 34 per cent. of 106 cases.

So that, taking fatal cases and cases that have been subjected to operation, a fæcal concretion is found to be the cause of the appendicular disease in 29 per cent. of our series, and in from 35 to 50 per cent. when a large number are taken.

It is equally important that we should know the frequency with which concretions are found in the bodies of those who have died from some cause other than disease of the appendix. It is exceedingly common to find in general post-mortem work fæcal pellets in the appendix, which are beginning to be shaped and moulded by its peristalsis. But their frequency is obviously misleading, unless only those cases are taken where some alteration of the mucous surface has already resulted from the pressure. Of such cases there were five in the hundred bodies which I examined (5 per cent.) verified by microscopical examination.

Ribbert (*loc. cit.*) found thirty-eight fæcal concretions in 400 autopsies (10 per cent.), but it is not apparent whether ulceration had commenced in these cases or not.

Foreign bodies.

There can be no doubt that foreign bodies other than fæcal concretions are by no means common in the appendix. Fitz found them in 12 per cent. of 152 cases, Matterstock in 12 per cent. of 169 cases, Ferguson[31] in 7·5 per cent. of 200 cases, Krafft in 4 per cent. of 106 cases. Of the sixty cases of fatal general peritonitis and perityphlitic abscess at St. Thomas's Hospital which I am reviewing, in none was a foreign body found. At the same time, some of the specimens of fæcal concretion had a very close resemblance to the cherry-stone or date-stone of popular romance. So hard, resilient, and shell-like are they sometimes, that they can be made to bound on the concrete floor of the post-mortem room.

The foreign bodies which have been found are necessarily a varied assortment, of a kind more curious than important. Among them may be mentioned beans, fig-seeds, tomato-seeds, grape-seeds, orange-pips, raisin-stones, cherry-stones, nutshells, pieces of bone, robin shot, a nail, a bristle, and a leaf. A pin has been known to perforate the wall of the appendix and lead to general peritonitis.

Sometimes such foreign bodies form the nucleus or take part in the formation of a fæcal concretion. A splinter of wood has been found in this way. Pins have been found in several cases. Wister records a case of general peritonitis due to perforation of the appendix by means of a plug of concrete matter having the size and shape of a date-stone, and composed of small seeds associated with concrete matter deposited in layers arranged in concentric circles. It is however the exception to find any trace of foreign material in a fæcal concretion.

(C) INFECTIVE APPENDICITIS

This third form of disease of the appendix is of frequent occurrence. It is a form which is almost of necessity followed by a virulent degree either of a general peritonitis or of the local suppurative form, and as might be supposed our knowledge of it is derived from cases that have proved fatal. Most of these cases, though not all, are accompanied by a necrosis or sloughing of part of the appendix-wall: and though this necrotic condition of the appendix has been described in a vast number of isolated instances under the terms "gangrene," "sphacelation," or "sloughing," for the past fifty years, so far as I know, no definite theory of its causation has been put forward. In the classification of the forms of disease of the appendix, previously drawn up, it was stated that this condition may ensue either upon a previous catarrhal inflammation or upon an ulcerative appendicitis. And this is certainly true, and it is through this fact that so little has been done to ascertain the exact pathological process which is in action. In fact, it has been the custom for the most part, where an appendix has been found after death necrosed and perforated, to suppose that this has been due to a concretion, whether such a body be discovered or not.

Now some of these cases may certainly be of this nature, and it may well be believed that a fæcal concretion may break up in pus or at any rate pass unnoticed. In some cases, however, which have occurred at St. Thomas's Hospital this cannot have been the case. As will appear from the subsequent description, in some cases the area of necrosis and the actual perforation of the appendix-wall are far too limited in extent to have allowed the escape of a concretion, and in these cases the lumen of the tube has uniformly been found to be free from fæcal matter. Such a condition at once raises a suspicion as to their origin, and suggests the action of some agent not hitherto considered. And, further, clear evidence will be adduced to show that this condition, called here "infective appendicitis," is capable of exciting a violent rapidly fatal peritonitis, and nevertheless no necrosis and no perforation visible with the naked eye or with the microscope need necessarily be present.

To state the position briefly, it will be maintained that an acute inflammation of the tissues of the appendix-wall is a form of appendicular disease which must be recognised as of common occurrence; that this form of appendicitis may result from either the catarrhal or the ulcerative form already described, but that it may arise independently of either; and that it is rightly called and distinguished by the name of "infective appendicitis," because it appears to be in all cases associated with and probably due to a bacterial invasion of the tissues of the appendix-wall, and because in all cases it leads to a virulent peritonitis with a poisoning effect upon the patient.

Infective appendicitis is the most common cause of acute general peritonitis, and, as will be shown later, it accounts for nearly half the cases of this kind, and is a more potent cause than even a fæcal concretion.

Although as a rule there is present in these cases a necrosis of some part of the appendix-wall, yet it is possible for the patient to die before this necrosis has set in. Whether there is necrosis or not, these cases of acute inflammation of the appendix-wall appear to be equally of bacterial origin, and they must be considered together for this reason. Both the existence of any necrosis and its extent seem to be only matters of time.

Naked-eye appearances.

In the first place must be considered the appearances seen with the naked eye. A young adult, who has never had a day's illness in his life, may be seized at any moment with sudden abdominal pain, rapidly develop all the signs of general peritonitis, and die in the course of three or four days. Such cases are common enough: they are described in post-mortem reports as being due to "sloughing of the appendix." There is found after death to be perforation of the appendix resulting from a more or less extensive necrosis of its wall.

Usually the necrosis involves a considerable area of the peritoneal surface. Sometimes an inch or more of the tube undergoes this death in a large part of its circumference.

Sometimes the process encircles the tube, so that at the post-mortem examination it is found in two separate pieces, or it may be that the proximal part only can be found. In a remarkable case recorded by Stephen Paget[32] the whole appendix was thus detached and floated out in the pus on opening a peri-typhlitic abscess.

Though the destruction, which involves the peritoneal coat, is thus usually extensive, it may be comparatively insignificant, and affect a very small area. Sometimes at one part of the tube there may be seen upon its peritoneal surface a dark patch of doubtful vitality, and in this patch may be one or two minute perforations, just capable of admitting a pin, or it may be, though it is a rare occurrence, that the appendix may present a small discoloured spot without anything suggestive of an actual perforation.

All these degrees of necrosis have occurred in St. Thomas's Hospital in the last four years, and evidence will be adduced to show that they are all essentially of the same nature.

Microscopical appearances.

As regards the microscopical appearances, the first point to be noted is that apparently in all cases there is a preceding loss of the protective epithelium of the mucous membrane, and that in those cases which follow a chronic catarrhal inflammation or an ulceration there is necessarily also a loss of the basement-membrane upon which that epithelium was seated. In other words, there exists in these cases a raw unprotected surface exposed to fæcal bacterial contamination.

Although this is the condition upon which this necrosis supervenes, it can be seen that it is not a question of the mere extension of gross disease from the mucous membrane to the other structures of the appendix-wall by continuity of tissue. Though the protective surface of the mucous membrane is thus deficient, yet the destructive necrotic process arises *ab initio* in the deeper structures of the wall. And this alone is suggestive of the penetration of some virulent agent into the wall. To show this clearly, it is best to take a specimen of moderate severity, where the necrosis is well marked and yet is not so extensive as to obscure all structural details.

The specimen shown in Fig. 10 was taken from the body of a boy aged six, who died after an illness of four days' duration from general suppurative peritonitis. The appendix was excised before death and the abdomen washed out, but the operation was undertaken too late to be of any avail. It was found to be two inches long, its wall was thickened and rigid, obliteration had nowhere occurred, and the lumen was quite free from fæcal matter. About half an inch of the middle of the appendix showed a grey discoloration occupying half the circumference of the tube, and this indicated the position and extent of the disease; but the actual visible lesion of its peritoneal coat was

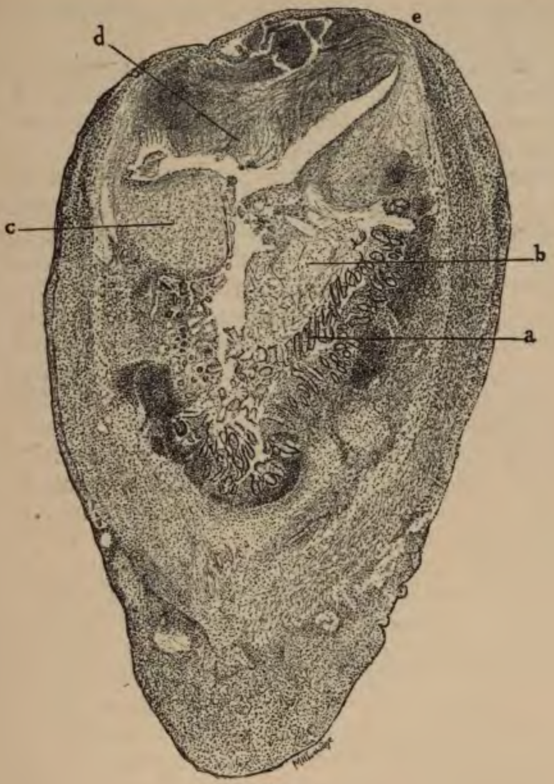

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FIG. 10.

Transverse section of an appendix, taken 1 mm. above a minute perforation which set up a fatal general peritonitis.

In the lower half of this figure the appendix-wall is nearly free from disease; in the upper half all the tissues between peritoneum and mucous membrane are acutely inflamed.

The glandular layer of the mucous membrane (*a*) of the upper half has been pushed down across the lumen into contact with the opposite surface by the pressure of an acute inflammation, which has arisen outside it, apparently in the deeper or lymphatic layer of the mucous membrane.

The general epithelial lining has been shed and the basement-membrane in part destroyed; but the changes in the glandular layer of the mucous membrane are quite out of proportion to the intense inflammation of the submucous and muscular coats.

a. Glandular layer of mucous membrane pressed down into contact with the opposite surface so that the lumen is obliterated.

b. Blood-clot in lymphatic layer of mucous membrane.

c. Mass of inflammatory exudation.

d. Necrosed muscular coats.

e. Point at which a pinhole perforation occurred 1 mm. below the level of this section.

limited to two minute perforations in the discoloured area, each of which would just admit a pin.

Now microscopical examination of this specimen (Fig. 10) shows that the discoloured area seen on the exterior of the tube was due to an accumulation of inflammatory products and a necrosis of the muscular coats at this spot. Further, the minute perforations lead through the peritoneal coat, not into the lumen of the appendix, but merely into this focus of softened, inflamed, and necrosed tissues in its wall. In other words, there is no perforation of the appendix in the ordinary sense. Finally, though there is some shedding of the epithelium at this point, the general structure of the mucous membrane is unaltered. The mischief clearly arose in the submucous areolar tissue, where free hæmorrhage has occurred, and the brunt of the destructive process lies still further outwards, in fact in the muscular coats immediately under the peritoneum. We have therefore in this case a small focus of acute inflammatory necrosis arising in the submucous and muscular coats, still separated from the lumen of the tube by a sound mucosa, but discharging itself outwardly into the general peritoneal cavity of the abdomen.

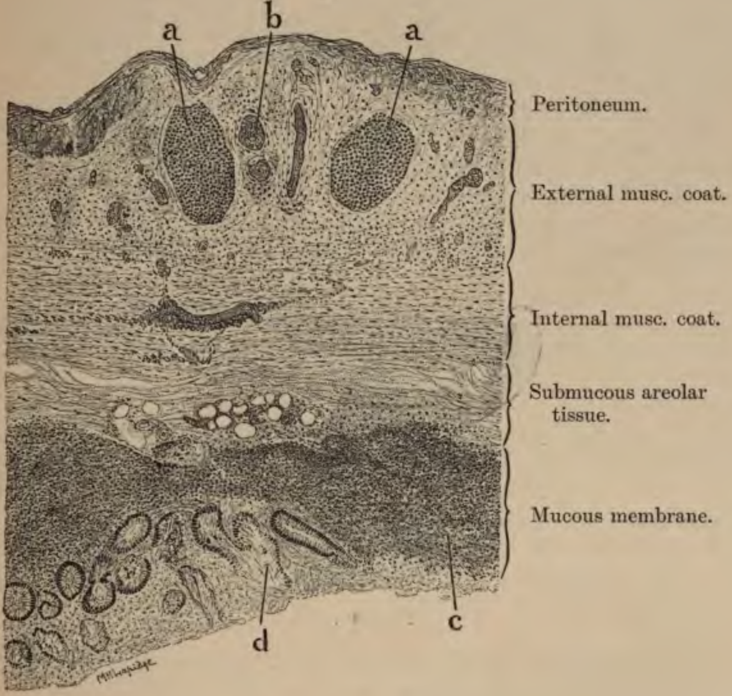
Many years ago Lebert[33] published a short note of three cases of acute peritonitis from disease of the appendix. He seems to have noticed that there was evidence of inflammation arising in the deeper structures of the appendix-wall. In all three cases there was a "diphtheritic inflammation" of the mucous membrane, and in one case there lay between the mucosa and the peritoneal coat some pea-sized abscesses, of which two had burst through into the lumen of the tube, while a third abscess was only prevented from rupturing externally by a thin layer of peritoneum.

The specimen shown in Fig. 11 points to the same conclusion as to the origination of the inflammation in the deeper tissues of the appendix-wall. It was taken from a boy aged 10, who died from acute suppurative peritonitis secondary to necrosis of the tip of the appendix. The mucous membrane was found to be diseased throughout. The section shown in the figure was taken near the cæcal end, about two inches therefore from the necrosed tip.

Besides the evidence of a chronic catarrh, there are seen to be hæmorrhages in and leucocytal infiltration of the submucous areolar tissue and of the muscular coats; and in the external muscular coat, immediately under the peritoneum, lie two small abscesses. The cause of the inflammation, whatever it was, in this case acted directly upon the muscular coat, and with our present ideas as to the causation of suppuration one can hardly doubt that these miliary abscesses were of bacterial origin.

One can see further how from the external rupture of such a small intra-muscular abscess a general infective peritonitis may be set up, while to the naked eye the appendix may present no sign of disease.

It has been already mentioned that in some cases of this kind there is no necrosis, but merely evidence of inflammation in the tissues of the appendix-wall. These cases, though not common, are accompanied by an equally virulent and fatal form of peritonitis.



× 40.

FIG. 11.

Transverse section of part of the wall near the cæcal end of an appendix, the tip of which had sloughed, setting up a fatal general peritonitis.

It shows chronic inflammation of the mucous membrane; there is no trace of epithelium or basement-membrane. There is an elongated collection of leucocytes around a distended vessel in the internal muscular coat, and two abscesses in the external muscular coat which have produced a bulging of the peritoneum over them.

a. Abscesses in external muscular coat.
b. Distended vessel.
c. Lymphatic tissue.
d. Remains of a crypt embedded in granulation-tissue.

As an example of this may be taken the appendix depicted in Fig. 12. The patient, a woman aged 41, had been taken suddenly ill with pain in the abdomen, vomiting, and diarrhœa, accompanied by shivering and profuse perspiration, and was admitted into hospital suffering from severe peritonitis. The operation was performed on the seventh day of illness, the appendix was removed, and the abdomen washed out, but without avail, for she died twelve hours later.

The part of the appendix excised at the operation is shown in Fig. 12 (I am indebted to Mr. Ballance for the use of the drawing). It was 1½ inch long, stiff and rigid. About the middle of its length was a discoloured area, having a longitudinal extent of half an inch and extending round one third of the circumference. On this area were seen two dark points, which were thought at the time to indicate minute perforations. In fact, the appearance was very similar to that of the appendix from which Fig. 10 was taken.

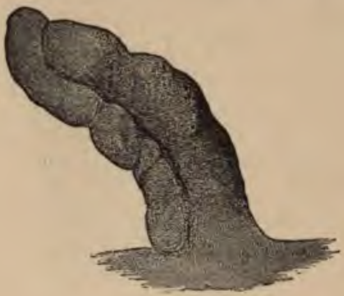


FIG. 12.

An appendix excised after it had set up an acute general peritonitis. The central half-inch showed some discoloration, but it was otherwise healthy in appearance, and it was not perforated.

On microscopical examination it was found that the thickening of the tube was due mainly to an intense œdema of the submucous areolar tissue, and partly to a layer of inflammatory exudation upon the peritoneal surface. Its lumen was very small, owing to the swelling of the submucous coat, and was quite free from fæcal matter. There did not appear to be any gross affection of the mucous membrane. The submucous areolar tissue showed great œdema, its wavy strands were loosely knit, it formed more than one half of the total thickness of the wall of the tube, and its arteries and veins were greatly distended. It showed also a moderate degree of leucocytal infiltration, and many large cells in its meshes having three or four times the size of a leucocyte. The muscular coats showed great vascular distension; their trabeculæ were split up and separated by hæmorrhages and collections of leucocytes. The fibrous substance of the peritoneal coat was transformed into an open meshwork consisting of coagulated fibrinous exudation, in which numerous cells, large and small, were entangled. And on the surface of the peritoneum was a layer of reticulated inflammatory exudation. There was no necrosis of any part of the wall, and no perforation. The appearance of the peritoneum and muscular coats is shown in Fig. 13.

In this case, then, a fatal general peritonitis was set up by an inflammation of the deep structures of the wall of the appendix, without any necrosis of tissue and without any perforation of the wall.

Three stages or forms of infective appendicitis.

These three specimens, above described, have been selected, as they seem to represent three stages or forms of an acute inflammation of the outer tissues of the appendix-wall. In the first the inflammation was accompanied by necrosis of the muscular coats; in the second, by suppuration in them; in the third, by their infiltration with leucocytes and inflammatory exudation. The first of these forms is by far the most common; it may arise at any moment in the course of a chronic catarrh of the tube, or in the ulceration of the interior of a cystic dilatation, or in the ulceration due to the presence of a fæcal concretion. The other two forms are less common, and may arise at any time in the course of a chronic catarrh, and even (as the specimen shown in Fig. 12 indicates) in cases where the microscope reveals very slight change in the mucous membrane. All three forms are alike followed by virulent peritonitis. In all three forms bacteria are found in the muscular and submucous coats, as will be subsequently described.

The bacterial origin of infective appendicitis.

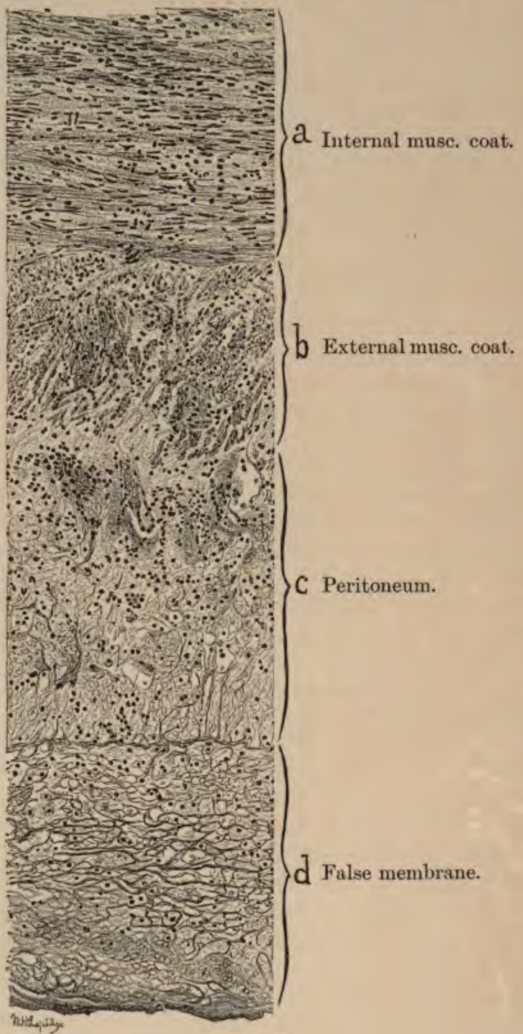
There is good reason for believing that this infective appendicitis is due to an invasion of the tissues of the appendix-wall by microbes of one or more kinds, which either normally, or at the time of the disease, are inhabiting the interior of the tube.

In the first place, the fact which has been established, that the inflammation arises in the deep tissues of the wall, is strongly suggestive of the action of some agent which has either penetrated to that spot from without, or has been brought thither by the blood-stream.

In the next place, a violent inflammation of this kind, able to lead to tissue-death or to abscess-formation in so short a space of time, is, so far as our knowledge goes, invariably of bacterial origin.

But the proof, if such it be, lies in the fact that in all such cases, so far as my observation goes, bacteria can be readily demonstrated actually in the tissues of the appendix-wall. On the other hand, in normal appendices no bacteria are to be found, except upon the free border of the epithelium lining the tube.

In the specimen from which Fig. 11 was taken the mucous surface is as usual swarming with bacteria, mostly rod-shaped. In the submucous tissue a few short thick rods can be seen,



× 100.

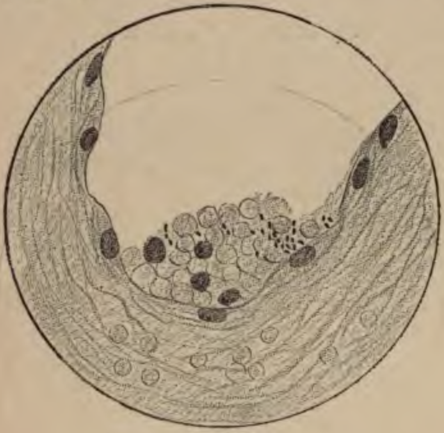
Fig. 13.

Transverse section through the discoloured area of the appendix shown in Fig. 12. (Only part of the thickness of the wall is represented.

It shows cellular infiltration and hæmorrhages in the muscular coats. The peritoneum is converted into a fibrinous meshwork, and on the peritoneum lies a layer of quasi-diphtheritic membrane.

and these become more numerous in the muscular coats. At some spots in the outer muscular coat, where are aggregations of leucocytes, the bacilli are thickly packed. They are for the most part short thick rods, sometimes so short as to approach to a coccus form, and in this case they are commonly arranged in pairs. They may be seen to lie also in the distended veins, and Fig. 14 represents a sketch of a clump of leucocytes with a few red corpuscles which was adherent to the inner wall of a small vein.

Again, the œdematous submucous tissue in the appendix, represented in Fig. 12, is swarming with similar bacilli. These are shown in Fig. 15.



× 500.

FIG. 14.

Short bacilli in a vein in the internal muscular coat of the appendix shown in Fig. 11.

So also in the specimen from which Fig. 10 was taken, the submucous and muscular coats show colonies of similar rods, as depicted in Fig. 16.

It is noteworthy that these bacilli are decolorised by Gram's solution. They are readily stained by carbol-fuchsin. Now, as will be subsequently detailed, in almost all cases of general peritonitis and of local perityphlitic abscess the bacillus coli communis is found in the exudate, and nearly always as a pure culture. This same bacillus is known to be a nearly constant inhabitant of the appendix. We know then of the presence of this bacillus in the lumen of the tube, and we find the same bacillus in the peritoneal exudate arising around and outside the tube; and the bacilli depicted above in the tissues of the appendix-wall have the superficial characters of the bacillus coli communis. So that thus far the evidence is in favour of the theory that this microbe has penetrated into the wall from the interior of the tube.

The presence of a bacillus in the tissues of the appendix-wall at once opens up the wide subject of the life-history of the microbes inhabiting the intestines.

Bacteriology of intestines.

The conditions in the intestines are very favourable for the growth of bacteria. Our knowledge of the various species is largely due to the work of Escherich in 1886. Many of them are pure saprophytes, but some are pathogenic. Some species are nearly constant, and some are found occasionally, as if they had been taken in with food and had escaped destruction in the stomach. Gillespie[34] has shown that pathogenic organisms which are usually very markedly affected by hydrochloric acid may pass through the stomach unharmed, if the meal with which they are ingested be large and chiefly proteid in character, especially when from any cause the supply of acid be somewhat deficient.

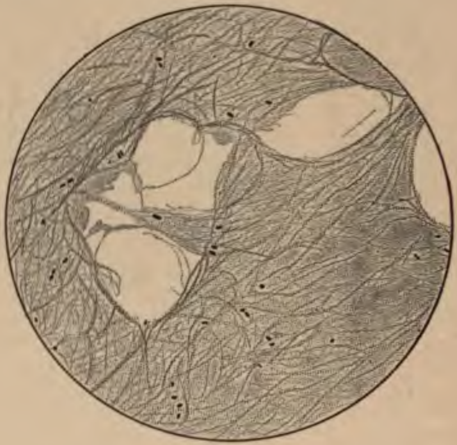


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FIG. 15.

A drawing made from a photograph of the submucosa of the appendix represented by Fig. 12. As many as twenty or thirty bacilli can be seen in a field lying in the œdematous tissue.

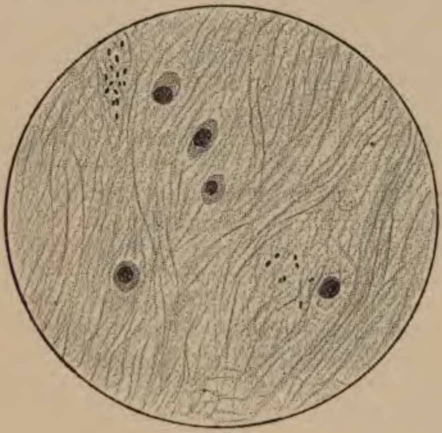
Bacteria are found to be far more numerous in the large than in the small intestine, and moreover it has been shown that they are more numerous in the intestines of carnivora and omnivora than in those of herbivora.

Some fifteen pathogenic forms have been found in the intestine, excluding the microbes associated with typhoid fever, tuberculosis, and cholera.

Increasing experience is tending to diminish rather than to amplify this list of pathogenic intestinal organisms, by establishing from time to time the identity of forms that had been previously described as distinct. But four forms stand out prominently as having a probable connection with disease in man—viz. bacillus coli communis, proteus vulgaris (and possibly proteus mirabilis), staphylococcus pyogenes aureus, and streptococcus pyogenes.

Bacillus coli communis in the appendix.

Of these forms, bacillus coli communis is so much more abundant than the others in the large intestine of man that in the majority of cases it is obtained from the mucous membrane as a pure culture. Especially is this the case in the vermiform appendix. Hodenpyl,[35] who was one of the first to suggest the connection of appendicitis with bacillus coli communis, found it to be present in the interior of five normal appendices and of fifteen appendices with internal inflammation. In nineteen of these twenty cases he obtained it as a pure culture; in the remaining case it was associated with streptococcus pyogenes. Hodenpyl confirmed the observation of Escherich, that in stillborn and new-born children the meconium is free from bacteria.



× 500.

FIG. 16.

Submucosa of the specimen from which Fig. 10 was taken, showing two colonies of bacilli.

While bearing in mind that such important organisms as staphylococcus pyogenes aureus and streptococcus pyogenes have been found in the appendix, though rarely, we may take it that bacillus coli communis is a nearly constant inhabitant of this part.

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Invasion of intestinal wall by bacteria.

It being the case, then, that the intestine, at any rate in part, is lined with microbes (three forms of which are known to be pathogenic in man), a question arises as to their effect upon the intestinal wall and as to a possible counter-effect of the intestinal wall upon these parasites. If we consider that they are lying upon the surface of the mucous membrane, separated only by a single layer of columnar cells from a subjacent tissue rich in lymphatics and capillaries, it is certainly remarkable that we have so few trustworthy examples of their passage into the general circulation.

As regards the alimentary canal, there is a fair suspicion that the tonsils may be a point at which microbes may penetrate to the deeper tissues and gain entrance into the circulation. The occasional occurrence of pylephlebitis and hepatic abscess in cases where no lesion whatever of the intestinal tract can be found may be adduced with some reason as evidence of a microbic invasion of the intestinal wall. Typhoid bacilli can be seen to invade the muscular coat of the bowel in the floor of the intestinal ulcers. Peritoneal tuberculosis is probably due to an infection from milk-borne bacilli in the intestine. And, finally, the general belief at this time is that in most of the specific fevers and in such general microbic diseases as malignant endocarditis and pyæmia without open wound, the entrance of the pathogenic organism is effected through the mucous membrane of the alimentary or pulmonary tract.

Yet if all this be granted as true, it still remains a matter of wonder, considering the constant presence of vast numbers of microbes in the intestines, that instances of their penetration into the healthy intestinal wall should be so rare.

The resisting power of the intestinal mucous membrane offers a wide field for investigation. The only explanation which can be offered at the present time is one that is grafted on Metschnikoff's theory of the struggle that ensues from time to time between invading bacteria and certain defensive cells of the host. If a pathogenic micrococcus be introduced beneath the skin accidentally or purposely, the struggle begins as shown by the phenomena of phagocytosis. As regards the intestinal wall, however, the struggle may be said to be continuous from the first day of a man's life to the last. Through the whole period it must be supposed that a defensive action is maintained by some elements of the intestinal wall; and this defensive action moreover is discriminating, for it allows the passage of fluids, substances in solution, and fat particles into the lacteals, while refusing admission to microbes. Such a purposeful defence as this can hardly be attributed to any element other than a living cell.

It is not necessary for the present purpose to go at length into this theory of Metschnikoff as to the protective part played by certain cells either as regards a local point of invasion or as regards the production of general immunity. But the observations of Ruffer[36] must be quoted as a most important extension of Metschnikoff's theory and as bearing directly on this question of an invasion of the vermiform appendix.

Ruffer's observations relate to the intestine of rabbits. He notes the innumerable forms and size of the micro-organisms lining the intestine. There are abundant leucocytes (which from their properties he calls "microphages") to be seen invading the superficial parts of the Peyer's patches, lying between the columnar epithelial cells even as high as their free border. So numerous are they sometimes as partly to obscure the epithelium. They are present also among the epithelial cells of the general lining of the intestine, but are far more numerous in the epithelium covering the Peyer's patches. These microphages contain in their substance numerous microbes, for the most part short but some of very large size. On the other hand, microbes are never found lying free between the epithelial cells, for when there they are always enclosed in a leucocyte. In all parts of the intestinal canal Ruffer has observed this process, but it takes place far more actively in some parts than in others. We must suppose that a greater activity in this microbe-destroying process implies not a greater activity on the part of the microphages but a greater need for their activity—in other words, a more vigorous invasion of the intestinal wall by the microbes. And therefore a special interest from our present point of view attaches to Ruffer's observation that "the Peyer's patch placed near the ileo-cæcal valve and the vermiform appendix are the structures in which these processes are most marked."

The description is completed by an account of the large mono-nucleated cells or "macrophages" which are to be found everywhere under the epithelium, but more abundantly in the deeper part of the lymphoid tissue. These contain numbers of bacilli in a more or less degenerate state, as well as dead and living leucocytes.

All these useful and suggestive observations of Ruffer's are amply supported by figures. So far as they go, they point to a fair and reasonable explanation of the resistance which is exercised by the intestinal wall against the penetration of the myriads of bacteria lying upon the mucous surface.

Very strong evidence of what may be called successful invasion of the walls of the appendix by the microbes contained in it is afforded by bacteriological examination of the peritoneal exudate around a diseased appendix. In all cases, as will be subsequently detailed, the bacillus coli communis is found in the serous and purulent fluids of appendicular peritonitis; and in nearly all cases it is found as a pure culture. As this is the very organism which abounds in the lumen of the tube, its universal presence in the peritoneal exudate around the appendix goes far to prove its passage from within outwards through the wall.

Evidence derived from other parts of the intestine.

There is moreover a considerable amount of evidence, pointing to the passage of this particular bacillus through the wall of the intestine when the vitality and resisting power of the intestine is impaired.

Clado[37] in 1889 found a bacillus in the sac of strangulated hernia in many cases, and this bacillus has been since identified with bacillus coli communis. Laruelle[38] found it under the same circumstances. Macaigne[39] made a similar observation. Tavel and Lanz[40] found it in the peritoneal exudate of three cases out of five of internal strangulation of the bowel.

Welch[41] has obtained pure cultures of it from cases of peritonitis which were due to ulceration of intestines without perforation.

Cornil[42] has shown the presence of bacteria in the substance of a necrosing intestinal wall.

Experimental evidence.

Further, Bonnecken[43] found microbes, for the most part bacillus coli communis, in every instance of numerous experiments in producing artificial strangulation in dogs. And the observation of Arnd[44] may be quoted on this point. In experimenting with bacillus pyocyaneus and bacillus prodigiosus he found that if a portion of the intestine of a rabbit be constricted by means of a rubber band, bacteria pass rapidly through the wall as soon as stasis has been induced in the blood-vessels of the constricted bowel.

An explanation of the particular liability of the appendix to bacterial invasion.

There is still a difficulty to be overcome before the view that the bacilli, which are associated with acute inflammation of the appendix-wall, can be accepted as the cause of that inflammation. How comes it that no similar acute inflammation with necrosis of tissue ever supervenes upon lesions ulcerative or otherwise in other parts of the intestinal tract? A breach of surface commonly occurs, for example in the colon in typhoid fever, and the same microbes, so far as our knowledge goes, inhabit the colon as the appendix, yet whenever peritonitis is excited by such disease of the colon it is through mechanical rupture of the floor of a deep ulcer, and not, as is so commonly the case in the appendix, through the intermediation of an acute inflammation of the whole thickness of the intestinal wall.

This difference between the appendix and other parts of the intestine must lie either in the structure of the part, or in the species of microbe inhabiting it, or in the nature of the initial lesion which exposes the deeper tissues to infection.

One can hardly doubt that it is in the structure of the part that the difference lies: not in the coarse structure as known in histology, for it cannot be supposed that the greater amount of lymphatic tissue or the different arrangement of the muscular coats can be sufficient to explain this proneness to bacterial invasion and inflammation on the part of the appendix, but rather in that subtle structure which determines the degree of resistance of a tissue to disease. One man differs from another in his power of resistance; the more degenerate the man, the less resistance can he exert. In like manner, one organ in a man differs from another. And in the appendix we are dealing with an organ which is degenerate and functionless from first to last, and its scanty power of resistance to bacterial invasion is but another way of expressing this fact.

The determining cause of bacterial invasion.

It has been shown how a chronic catarrh and also ulceration by the pressure of a fæcal concretion may exist apparently for a considerable time, and yet, though the epithelial protection has been removed, no bacterial inflammation of the appendix-wall need occur. As regards a concretion, it may be found in the post-mortem room to be held in an appendix whose wall at this spot consists merely of thinned-out muscle and peritoneum, and yet there may be no appearance of past or present peritonitis. This apparent rarity of a bacterial invasion under these circumstances need not be considered as in any way militating against the theory here advanced. The same fitfulness in bacterial action is seen in every disease which is of microbic origin. It is hardly necessary to give examples. It is seen in the case of every specific fever. It is in full accordance with our present knowledge to consider that a microbic disease is not to be regarded as a sudden new irruption of micro-organisms, but rather as the weakening of the resisting force in a struggle which is of lifelong duration.

As regards the particular instance of the appendix, doubtless the speedy obliteration of the lumen by union of its walls is an important conservative process, and probably the formation of a layer of true granulation-tissue is to some extent protective. It is possible also that the lymphatic layer has some action in this direction.

But a more important factor is probably the varying virulence of bacteria under different conditions. This particular organism, bacillus coli communis, is particularly variable in this respect. It may retain its virulence in artificial media for several months, but on agar it loses it more quickly than in broth, and cultures may be by this means attenuated until virulence is lost (Roswell Park).[45]

Ekehorn[46] has found in experimenting upon animals that the bacillus coli communis obtained from mild forms of appendicular disease is much less virulent than that from acute severe cases.

And, further, Macaigne[47] has shown that a culture taken from a healthy intestine is harmless when injected into the peritoneal cavity.

Again, there are other factors, in the action of microbes, which have been suggested by numerous observations. There is the possibility of conjoined action of different species in producing disease. Moreover, the observation of Cheyne on the action of proteus vulgaris bears on the point. He found that $\frac{1}{10}$ c.c. of a broth culture of this organism injected into the tissues of a rabbit invariably caused death in 48 hours, while the animal recovered from $\frac{1}{40}$ c.c. If however $\frac{1}{40}$ c.c. was injected with the addition of 1 c.c. of a sterilised culture, death was invariable. Yet the sterilised culture by itself produced no effect. The explanation suggested by him was that the toxic products present in the sterilised culture overcame the natural resisting powers of the tissues, and the living proteus vulgaris was thus enabled to grow and multiply to an extent sufficient to cause death.

It is impossible, then, at present to determine why in one case more than another the bacilli are enabled to penetrate the appendix-wall. It may depend on a varying virulence of the organism or on the possible presence of chemical substances enabling it to act with increased vigour or on a lowering of the tissue-resistance from this or some other cause. But inasmuch as a like apparent capriciousness is seen in all microbic diseases, it has no weight in the particular instance of the appendix.

Relation of the necrosis to the bacteria present.

As has been already stated, these cases of acute inflammation of the appendix-wall are for the most part associated with some degree of necrosis of its tissues. Some observers have been inclined to regard this acute necrosis as being due to a primary thrombosis of a large arterial branch, and the inflammation as secondary to the necrosis. Fowler[48] indeed narrates a case which may be of this nature. He says "it may happen that but a single nutrient artery exists, and that this may not give off any branches before reaching the tip of the appendix, being there reflected upon itself in the submucosa. This condition was found to exist in one case: a stasis-thrombosis of septic origin occupied the lumen of the vessel at a point about one-third of the distance which the vessel traversed from the tip to the base of the organ. Under these circumstances the primary inflammatory lesion occurs in the peripheral portion of the organ, and the occlusion of the vessel takes place at this point: following this, a gangrenous condition will supervene in the portion of the appendix lying between the point of occlusion and the base, since there is no other blood-supply save through the reflected trunk."

There is certainly nothing improbable in Fowler's explanation of the particular case to which he alludes, but such a gangrene by occlusion of the main artery or of a large branch must be of very rare occurrence. In the first place, the main artery, a branch of the ileo-colic, runs in the mesentery of the appendix giving off branches at regular intervals. The anastomosis in the submucosa is so free that no occlusion of any one branch of the main artery could have any harmful effect. In the second place, even if the main artery were occluded at its origin, it is doubtful whether a total necrosis would ensue, for the submucosal vessels anastomose with those of the cæcum.

It is moreover difficult to see how from any primary inflammation of the interior of the appendix such occlusion could be produced in the main artery, running as it does remotely in the mesentery.

The appearances seen in Fig. 10 cannot be explained as resulting from arterial thrombosis: the necrosis occupies too limited an area, barely half of the circumference of the tube. And this specimen may be taken as the type of the condition usually found.

Finally, in none of my specimens could any trace of arterial thrombosis be discovered by microscopical examination, much less of any of the endarteritis that has been from time to time described. All arteries and arterioles are uniformly healthy, but distended with red blood. Nor can the necrosis be ascribed to the mere pressure of a concretion upon the wall. For in the first place, as has been already shown, such necrosis occurs independently of any concretion, and, in the next place, it will be noted that the necrosis and perforation which may occur in conjunction with a concretion do not always correspond to the actual bed in which the concretion rests, but are sometimes above it and sometimes below it.

I am inclined to attribute the necrosis either to the direct action upon the tissues of the bacilli with which they are swarming, or to the effect of the toxic products of bacillary life. But in cases where a large portion of the appendix undergoes necrosis it is possible that it results in part from the strangulation of the capillaries in the affected area, and that it is analogous to the sloughing of skin from a tense œdema, and to the sloughing of tendons in whitlow.

General conclusions.

The affections, then, of the appendix which are demonstrated by actual direct observation as being the basis of a local or a general appendicular peritonitis may be thus summarised :—

(1) Catarrhal appendicitis, marked particularly by shedding of the protective epithelium, apt to pass into a chronic condition with thickening of the wall.

(2) The cystic condition of the appendix, whether due to a post-catarrhal stricture or to kinking.

(3) Ulcerative appendicitis due to the presence of a fæcal concretion or a foreign body.

(4) Acute infective inflammation of the wall of the appendix, commonly associated with necrosis of its tissues; this may occur when the mucous membrane shows slight signs of chronic

disease, but in the majority of cases it supervenes upon one of the foregoing conditions.

It is made clear both by clinical and by post-mortem observation that the first three of these morbid conditions are not attended by symptoms until the peritoneal coat of the appendix is involved. It is possible that vague sensations of uneasiness may arise before this point is reached, but this general truth is, I believe, beyond controversy, that in all cases which claim medical attention the peritoneum is already inflamed.

The fourth condition necessarily entails the immediate onset of severe symptoms.

(D) OTHER MORBID CONDITIONS OF THE APPENDIX

Tubercular disease of the appendix.

In cases of tubercular ulceration of the bowels, it is exceedingly common to find a definite tubercular ulcer in the appendix. Sometimes it appears that the lymphoid tissue in its wall undergoes at one or more spots a chronic inflammation such as is produced by tubercular infection in similar tissue elsewhere, and in such a case one or more caseous nodules may be found to lie in the submucosa.

It is doubtful, however, if the condition described here as perityphlitis ever arises from this cause. A general peritonitis may be set up by perforation of a tubercular ulcer in the appendix, as from a similar ulcer in any other part of the intestine, but even this is a rare occurrence.

Typhoid ulceration of the appendix.

Similarly, the appendix may present the lesions of typhoid fever, and a general peritonitis may be set up by its perforation from this cause. But it is clear that such lesions play no part in the causation of the local peritonitis which is more particularly under consideration.

Actinomycosis of the appendix.

So far as I know, only one certain case of actinomycosis of the appendix has been recorded. This was published by Ransom in the *Med. Chir. Trans.* 1892. The disease of the appendix was accompanied by a retro-cæcal abscess, with which its cavity communicated, and the patient succumbed with a metastatic actinomycotic abscess in the liver after nine months' illness. The great interest in the case lies in the fact that at the onset, and for a considerable time afterwards, there was nothing to suggest anything unusual. It appeared to be an ordinary case of perityphlitic abscess.

The possibility of such a condition as this must then be kept in view, whenever the course of the disease is unduly prolonged.

CHAPTER V

Relation of these morbid conditions to the various forms of Peritonitis.—General peritonitis, perityphlitic abscess, simple perityphlitis.
Causes of Disease of the Appendix.—Age, sex, season, occupation, anatomical and developmental considerations.

THE RELATION OF THESE MORBID CONDITIONS OF THE APPENDIX TO PERITONITIS

GIVEN one or other of the morbid conditions thus described, the bearer of the diseased appendix may be at any moment made aware that the peritoneum is attacked.

He is seized with sudden abdominal pain, and develops one by one the symptoms that will be subsequently detailed. It may be that, owing to the sudden and profuse escape from the appendix of bacteria-laden material, which is carried swiftly by the movements of the intestines to all parts of the abdomen, a general peritonitis is at once established beyond recall, or possibly, and more fortunately, owing to a less virulent poison, the effect is more limited, and the peritoneum covering the cæcum and the coils of small bowel which happen for the moment to lie in its neighbourhood is alone affected. An exudation appears on the peritoneal surface of these parts, which at once coagulates, and the mobile intestines are thus caught and fixed into a tolerably firm mass, which may be readily felt with the hand. Such a mass does usually undergo resolution, the inflammatory products being for the most part absorbed, and to a slight extent replaced by organised tissue-adhesions. But around the diseased appendix, still lying in the centre of the mass, there may arise, by an increase either in the amount or in the virulence of the poison, a pyogenic inflammation; and drop by drop pus may accumulate around it, exerting pressure on the surrounding structures, until in the second week of illness a localised abscess is formed, securely walled in by agglutinated intestine.

Thus peritonitis of appendicular origin presents itself clinically in three forms or degrees:—

(1) Acute general peritonitis.

(2) Peritonitis, either limited from the first to the right iliac fossa and its neighbourhood, or becoming so limited in the course of the illness, and ending in the formation of pus (perityphlitic abscess).

(3) A local adhesive peritonitis limited to the right iliac fossa associated with the exudation of a coagulable material, which is capable of undergoing absorption or organisation (perityphlitis).

These forms will be subsequently considered from their clinical aspect, but the present purpose is to show the connection between them and the known morbid conditions of the appendix, which have been described in the previous chapter.

(A) GENERAL PERITONITIS

Our knowledge of the conditions of the appendix which give rise to acute general peritonitis is fairly complete, for the reason that abundant opportunity is afforded for post-mortem observation.

Fæcal concretion.

Of the twenty-seven fatal cases in our series in which a post-mortem examination was made, the cause in eleven was the presence of a fæcal concretion. It is not necessary to add much to the account already given of the effect produced by this means. It can be seen how by a continuance of the process, illustrated by Fig. 9, especially if any sudden movement takes place or violence be applied, rupture of the attenuated appendix-wall may readily ensue, with subsequent escape of fæcal matter and bacteria into the peritoneal cavity.

In such cases the concretion is sometimes found free in the peritoneal cavity, while a ragged rent in the appendix indicates its mode of exit; or the rent is found in the appendix, with the concretion still firmly held in its original bed.

A general peritonitis will result as from perforation of other parts of the intestinal canal.

In a considerable number of cases the origin of general peritonitis from the impaction of a fæcal mass is not of such a mechanical nature. There may be no actual breach of continuity in the appendix-wall. In these cases the portion of the wall upon which the concretion rests is found to be black, soft, and necrosed, and it has in fact undergone the acute necrotic inflammation which has been already described. The concretion here acts by destroying the protecting mucous membrane and by laying bare the deeper tissues of the wall.

It is probable that peritonitis arises from a foreign body in the same way as from a concretion.

Cystic appendix.

Of our twenty-seven fatal cases of general peritonitis a cystic appendix was the cause in three. It is not difficult to understand the sequence of events. The inner surface in these cases, especially where the stricture has arisen from a chronic catarrh, is commonly ulcerated, either uniformly or in one or more separate spots. The condition of tension, in which the appendix-wall must be, is calculated to insure a progressive ulceration, until the peritoneum is involved or some sudden strain or movement produces a rupture in the floor of an ulcer. Two such cases have been already described.

Fig. 17 shows a stricture in longitudinal section with ulceration of the mucous membrane on the distal side of it.

In these cases it is not uncommon to obtain a history of some sudden exertion as the starting-point of the symptoms. The immediate cause of the peritonitis is commonly thus the perforation or rupture of an ulcer, but in some instances the final change is the same acute necrosis of the whole thickness of the appendix-wall which has been described as occurring in the case of a concretion.

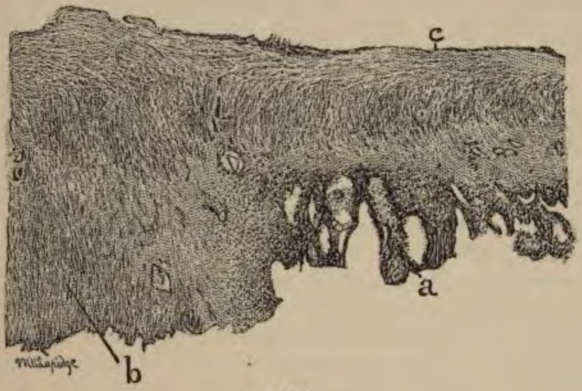

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FIG. 17.

Longitudinal section of an appendix, perforation of which set up a fatal peritonitis.

a. **Ulcerated mucous membrane.**
b. **Stricture (total replacement of mucous membrane by fibrous tissue).**
c. **Peritoneum.**

Infective appendicitis.

An acute inflammation of the appendix-wall, usually associated with necrosis of some part of its structure, may thus end the scene of the concretion and of the cystic appendix. But it may also arise as the substantive condition. In thirteen of our twenty-seven fatal cases of general peritonitis the cause was found to be of this nature, without any concretion and apart from any cystic condition. In all the specimens examined there was evidence of previous catarrh of the mucous membrane. The onset of general peritonitis and its virulence in these cases are explained by the view that the wall of the appendix is itself infected by bacteria.

(B) PERITYPHLITIC ABSCESS

Sufficient material has accumulated to give a general idea of the changes in the appendix which are associated with local intraperitoneal abscess around it. Our knowledge however is less precise than in the case of general peritonitis, because the condition is rarely fatal, and, although all such cases are (or should be) subjected to operation, there is a general opinion (at any rate in this country) that it is not wise to examine too closely into the condition of the appendix, and indeed in many cases such an examination is not possible at the time of operation. In the first place, it is clear that of the four conditions of the appendix which form our starting-point, two tower above the others as being the cause of this local abscess—viz. a chronic catarrh followed by acute inflammatory necrosis of the wall, and the ulceration due to a concretion.

Of twenty-seven cases from all sources, of which a sufficiently clear description can be obtained, it appears that ten were due to the former and fifteen to the latter. Of the remaining two, one was due to a simple chronic catarrh without necrosis, and the other to a cystic appendix. And it is beyond doubt that an abscess may form around an appendix which has no perforation of its wall.

It is probable that these figures, small though they be, give us a rough idea of the truth. But it may be that the relative number of cases due to a concretion is higher than would appear from this collection of cases; for it must be remembered that at any rate in this country it is the custom merely to open the abscess and do no more, so that it is always possible that a concretion lying in the pus partly disintegrated may be washed out and so escape notice.

In four of the fifteen cases of concretion, the actual cause of the peritonitis was necrosis occurring in the part of the wall upon which the concretion rested.

A case in which a perityphlitic abscess resulted from a chronic catarrh without perforation or necrosis has been recorded by Fitz.[49] The patient had several attacks of local peritonitis, from which recovery took place in the usual way: in a subsequent attack an abscess formed and was drained. After recovery, another attack occurred, and the appendix was excised. It was found to be thickened by chronic inflammation, but neither ulcerated nor perforated.

(C) LOCAL ADHESIVE PERITONITIS

As regards this local peritonitis, which is not followed by the formation of pus, we are compelled to draw our information almost entirely from the results obtained by operation. The American records contain many illustrations. This form may be said to have in itself no tendency to death, and it is for this reason that it has been so long misunderstood.

As might have been expected, the condition of the appendix here stands in contrast with the condition met with in the more severe forms of peritonitis already considered. Perforation from any cause and the infective form of inflammation fall into the background. Of twenty-five cases of which I can find a clear account, fourteen were due to simple chronic catarrh without necrosis, seven to the cystic condition, and four to the presence of a concretion without perforation.

Considering the great frequency of this local peritonitis, these figures form a somewhat slender foundation for any large conclusion, but they are so fully in accord with what we should have expected from general pathological knowledge that I believe they may be taken as a true index of the conditions commonly present.

General conclusion.

It appears, then, that we are justified in concluding (1) that a general peritonitis and a perityphlitic abscess depend mainly on the presence of a concretion or on an acute infective inflammation of the appendix-wall usually associated with necrosis of some part of it; (2) that the far more common form of local adhesive non-suppurative peritonitis has for its basis a chronic catarrh or a cystic condition of the tube. These conclusions are in agreement with the view that at any rate the more acute forms of peritonitis are invariably due to the entrance of bacteria into the peritoneal cavity.

THE CAUSES OF DISEASE OF THE APPENDIX

It is difficult to make a sharp distinction between the etiology of appendix-disease and that of the resultant peritonitis, by which alone it can be recognised. The actual exciting causes of the various forms of peritonitis will be mentioned later.

The incidence of appendicular peritonitis as regards age, sex, time of year, and occupation may be given here, although it is by no means certain that these factors have any causal connection with the primary disease in the appendix. We recognise appendicitis only by its having caused peritonitis.

Age. An important point is the narrow limits of the age at which the different forms of appendicular peritonitis occur. Any of these forms may occur at any age. There may be an attack in the first year of life or at the age of 76. But the subjoined table shows that the weight of the disease falls in early adult life. In 224 cases occurring at St. Thomas's Hospital—

From 5 to 10 there were 26 or 11·6 per cent.
,, 10 to 20 there were 97 or 43·3 ,,
,, 20 to 30 there were 67 or 30·0 ,,
,, 30 to 40 there were 18 or 8·0 ,,
,, 40 to 50 there were 14 or 6·2 ,,
over 50 there were 2 or ·9 ,,

Sex. The great preponderance in males is a known quantity. Of 224 patients, 161 were males and 63 were females.

Season. It seemed to me, while living in hospital, that this disease was particularly common in the summer, and there is certainly some difference in this respect apparent from the hospital records.

Of these 224 patients, 134 were admitted in the six summer months as compared with 90 in the winter.

Occupation. It is not likely that occupation has any bearing on the development of disease of the appendix. It attacks every one, from a sailor to a clerk, from an athlete to a sedentary tailor.

When disease of the appendix is once established, it appears that some slight influence is exercised by the daily occupation upon the occurrence of peritonitis. Attacks are found to be rather more frequent among such men as porters and carmen than in those engaged in less laborious work. They are also very common in young men who stand behind a counter for some eight or ten hours a day.

Anatomical considerations. The points in the anatomy of the appendix which throw light on the causation of its diseases have been already given.

It has been shown by Ribbert that its absolute length is greatest in early adult life, after which it tends to become shorter.

Bryant has shown that the lumen is larger in the male than in the female, that the larger the lumen the greater is the frequency with which it is found to contain fæcal or other matters, and that consequently fæcal or other contents are far more common in the male than in the female.

The bearing of these observations on the facts already given as to the age and sex most commonly affected is very clear.

Developmental history. Finally, there remains the fact that the appendix is obsolete and functionless, and herein lies its proneness to disease upon slight provocation.

CHAPTER VI

Causation of Peritonitis in general.
Peritonitis with a purulent exudation—(*a*) due to infection from without; (*b*) due to infection from within. Tuberculous Peritonitis, and idiopathic Peritonitis. Bacillus coli communis, and other Bacteria.
Peritonitis with a coagulable exudation.

THE ACTUAL CAUSATION OF PERITONITIS

It is becoming more and more evident that in all cases of peritonitis, associated with a purulent exudation, bacteria are to be found in the peritoneal cavity. The constancy of the species of bacterium found and the results of experiments afford strong evidence that the bacterium is the cause of the peritonitis, either *per se* or by means of the toxic products of its life.

On the other hand, as regards that variety of peritonitis which is marked by the production of a coagulable exudate, a bacterial origin is by no means so certain. This comparative uncertainty is due to the scanty opportunities available for examination, and its elucidation will be a gradual process.

(A) PERITONITIS ACCOMPANIED BY A NON-COAGULABLE PURULENT EXUDATION (ACUTE GENERAL PERITONITIS AND PERITYPHLITIC ABSCESS)

A large amount of work has been expended upon the bacteriology of peritonitis in the last five years, culminating with the work of Tavel and Lanz in 1893.

It has been shown on all sides that mechanical irritants introduced into the abdomen experimentally do not cause peritonitis, if they are absolutely free from micro-organisms. Powdered glass has no effect, but the particles become encysted (Sternberg); [50] a pellet of sterilised gauze placed in the abdomen has a similar peaceful end (Jalaguier).[51] Even sterilised fæcal matter may be introduced harmlessly into the abdominal cavity.

On the other hand, intense irritants can produce a peritonitis even apart from the agency of bacteria. It may be induced in rabbits and guinea-pigs by injecting into the peritoneal cavity such caustic fluids as strong mineral acids, acetic acid, nitrate of silver, and phenol (Pernice).[52] Sterilised croton oil has a similar effect. The possibility under certain circumstances (entailing damage to the bowel) of the passage of bacteria outwards through the bowel wall into the peritoneal cavity has been already discussed, and the observations of Clado and of Arnd have been already cited on this point. And it may be a question how far the peritonitis excited by powerful irritants may be really of bacterial origin, the damage done to the intestinal wall by the injected irritant leading to a bacterial invasion at that point. An observation of Fränkel[53] points in this direction. He found that the exudate produced by the injection of an irritant into the peritoneal cavity was at first sterile, but that at a later stage when it became purulent it was rich in bacteria.

However this may be with the case of powerful irritants, it may now be accepted that all cases of peritonitis in man, associated with a purulent exudation, are due to the presence of bacteria or their poisons in the peritoneal cavity.

At the same time, evidence is accumulating in favour of different forms of bacteria as the causes of the different clinical forms of peritonitis. An investigation into the bacteria of peritonitis is one not suited for experimental work, except as a means of confirmation. The first step can only be taken through cultivations made from the exudate of various forms of peritonitis. The chief workers in this field have been Fränkel, Bumm, Laruelle, Malvoz, Hodenpyl, Welch, Tavel and Lanz.

Bacteria associated with peritonitis.

Fränkel,[53] in 31 cases of peritonitis, found a pure culture of bac. coli communis in 9, of streptococcus in 7, of bac. lactis aerogenes in 2, of micrococcus pneumoniæ crouposæ in 1, and of staph. pyogenes aureus in 1. In 3 cases bac. coli communis was present in association with other bacilli, and in 4 cases the examination gave a negative result. In all of the 9 cases in which bac. coli communis was found, intestinal lesions were present. Since this publication, the bacillus described as bac. lactis aerogenes has been identified as bac. coli communis with slight modification.

Tavel and Lanz (*loc. cit.*) obtained similar results. In 59 cases they found :—

B. coli communis	—alone in	15	—in association in	16
Streptococcus	,,	3	,,	15
Staphylococcus	,,	2	,,	6
Pneumococcus	,,	0	,,	2
		20		39

In puerperal peritonitis it is found that streptococcus pyogenes is constantly present. This form with staphylococcus pyogenes aureus and albus are commonly found in peritonitis consecutive to laparotomy.

It may be said provisionally that from a bacteriological point of view all cases of peritonitis fall under one of these two heads: (1) due to infection from without; (2) due to infection from the intestine or the hollow viscera. A few examples of peritonitis cannot yet be included under these heads, but this is not due to opposing evidence but to want of opportunity for investigation.

(1) *Peritonitis due to infection from without.*

The first class of peritonitis needs no special description here. As regards puerperal peritonitis, peritonitis following laparotomy (with certain exceptions to be mentioned later), and peritonitis resulting from disease of the pelvic organs in the female, all observations agree that the bacteria present in the exudate are the pus-producing micrococci, streptococcus pyogenes in particular, often associated with staphylococcus. This fact is established by the work of Bumm, Fränkel, Tavel, and Lanz. Hodenpyl (*loc. cit.*) has recently produced the same results. He found staphylococcus pyogenes aureus in three cases of peritonitis with suppurative salpingitis and streptococcus pyogenes in another case of the same nature.

(2) *Peritonitis due to infection from intestine or hollow viscera,* (*a*) *with perforation;*

In the next place must be considered the case of peritonitis arising from a perforative lesion of the intestinal canal. The constancy of the presence of bac. coli communis in the exudate of such cases is beyond doubt, and goes far to justify us in looking upon this organism as the immediate cause of the peritonitis.

It is found as a constant inhabitant of the whole of the intestinal tract. Harmless enough probably in its native place, it can certainly assume a high degree of malignancy, as has been already shown.

Its frequency in cases of perforative peritonitis appears from the tables of Fränkel and Tavel and Lanz already quoted. Welch[54] puts forward the view that when this organism is found in the exudate of a peritonitis, the cause of the peritonitis is an intestinal lesion. Hodenpyl (*loc. cit.*) obtained a pure culture of it from a case of perforation of the stomach and from a case of perforation of the bowel.

Barbacci[55] found it in pure culture from six cases of general peritonitis due to perforation of the bowel. He has found it also in experimental perforative peritonitis in dogs.

Malvoz[56] obtained a similar result in the same class of case.

Of three cases of peritonitis resulting from suppurative inflammation of the gall-bladder, Tavel and Lanz found this bacillus in two, and pyogenic cocci in the third.

We are consequently in a position to say that in a vast majority of cases of peritonitis due to intestinal perforation bac. coli communis is found in the exudate, and as a rule in a pure culture.

(b) without perforation.

It has already been shown that there is good reason to believe that bacteria may pass through the intestine into the peritoneal cavity, when the intestinal wall is from some cause damaged, and yet not perforated. And there is further reason to believe that peritonitis, general or local, may be thus produced, even when the intestine presents but slight signs of damage.

The experiments of Arnd with bac. pyocyaneus and bac. prodigiosus, already quoted, may be cited as evidence. The observations of Clado, Laruelle, Macaigne, Bonnecken, Tavel, and Lanz on the presence of bac. coli communis in hernial sacs and on the peritoneal surface of strangulated bowel point in this direction.

Welch (*loc. cit.*) has found this same bacillus as a pure culture in peritonitis due to ulceration of intestines without perforation.

The suggestion has been recently made, that in some of the cases of peritonitis following laparotomy the peritoneal infection may arise not from air-borne bacteria but from intestinal bacteria which have passed through the intestinal wall. Careful bacteriological examination is necessary before this auto-infection can be established as a truth, but probably all surgeons can recall instances of peritonitis after abdominal operations where every care has been exercised to prevent aërial and cutaneous infection.

In some such cases, at any rate, the bac. coli communis has been found to be present. Welch[57] speaks of having found it several times as a pure culture in laparotomy wounds treated aseptically.

The possibility of a peritonitis due to this bacillus, resulting from rough handling of the bowel during operation or from the tearing of adhesions, is a question full of interest, but not ripe for discussion.

Tuberculous peritonitis.

In analysing and classifying a large number of cases of tuberculosis of the peritoneum (St. Thomas's Hospital Reports, vol. 20) I was impressed with the wide variation in the malignancy and virulence of this affection. Though all microbes may doubtless vary in virulence, the bac. tuberculosis among them, and though the inherited power of resistance of the infected tissue may certainly vary in degree, yet neither of these elements of variation seem quite sufficient to account for the wide limits met with in tuberculous peritonitis. In one case a patient may present scarcely any indication of illness, and the tuberculous infection may be discovered accidentally during an exploratory operation. In another case a patient may be suddenly seized with severe general and abdominal symptoms, so severe and sudden that a suspicion of perforative disease of the bowel is at once aroused, and he may die in three or four weeks, with an acute purulent peritonitis, presenting just the toxæmic condition which is seen in perforative peritonitis.

The possibility that such an extreme difference in malignancy might be due to other bacteria present in the peritoneal cavity alongside of the tubercle-bacillus was an obvious idea. The death of the patient in such acute cases, presenting the same symptoms of poisoning that are seen in perforative peritonitis, suggested such an explanation.

I have had but one opportunity of examining such a case.

The patient was a girl, E. B., a general servant, aged nineteen. About three weeks before admission into St. Thomas's Hospital she was suddenly seized with shivering and swelling of the abdomen: she continued her work for a day or two, but was then compelled to desist by increasing pain in the abdomen and a tendency to vomit. For the first three days after her admission into hospital she was extremely ill, restless and yet listless, with pinched face and sunken eyes, a temperature ranging from 101 to 104, and a rapid, feeble pulse. The abdomen was greatly distended, but not very tender. Respiration was entirely thoracic. The bowels were loose, the motions being light yellow, quite fluid, and very offensive. The tongue was furred and dry. It seemed to be a case of acute tuberculosis of the peritoneum, for the reason that there was a tendency to the induration and blush around the umbilicus that is so common in such cases, though the possibility of enteric fever could hardly be entirely excluded. In a few days, however, free fluid appeared in the flanks, and friction could be heard in the right axilla. The abdomen was opened, notwithstanding the great intestinal distension, thirty-five ounces of purulent fluid were withdrawn, and the peritoneal cavity was washed out as thoroughly as the condition would permit. Great improvement followed, but it was not lasting, and she died in less than five weeks from the beginning of the illness.

Soon after death cultivations were made with every precaution from the peritoneal cavity in two spots—viz. below the right and left costal margins, and both these gave pure cultures of staphylococcus pyogenes aureus.

At the post-mortem, the whole peritoneum, visceral and parietal, was found to be thickly studded with miliary tubercle: there was also a small patch of tubercle on the lateral surface of the lower lobe of the right lung, but none elsewhere in the body. The mucous membrane of the small intestine was hyperæmic and covered with mucus, but it showed no tubercle or ulceration.

Such a case as this may be taken to suggest the possibility that in some cases of simple tuberculous peritonitis the bowel-wall may be so altered as to allow of the escape of a pyogenic microbe and a consequent secondary and more serious infection of the peritoneal cavity.

So-called idiopathic peritonitis.

This possible migration of bacteria through the bowel-wall may also be subsequently found to explain those cases of acute general peritonitis for which no cause is discovered upon careful post-mortem examination.

Weichselbaum has observed two cases of primary peritonitis and pleurisy which he attributes to the micrococcus pneumoniæ crouposæ found in the exudate.

Almost every year such cases are met with in St. Thomas's Hospital, and though they may occur at any age they are most common, so far as my experience goes, in children and young adults.

If this bacterial invasion be a true story, the appendix, as being the part of the intestine having the greatest proneness to disease, should at any rate in some cases be found to be the site of the invasion. I believe this to have been the case in the two following instances.

A child died in this hospital in 1892 from general peritonitis. The abdomen was explored during life, and the appendix was examined and considered to be healthy. At the post-mortem no cause for the peritonitis was found. With the idea of a possible connection of such cases with disease of the appendix, I subsequently examined it microscopically. It showed complete loss of the epithelial lining and partial destruction of the basement-membrane, with inflammatory foci in the submucous areolar tissue and in both muscular coats.

In another case of similar unexplained peritonitis, the appendix showed no naked-eye sign of disease, though at one spot on the mucous surface I thought I could see a little discoloration. On examining this spot microscopically there was seen to be complete destruction of the epithelial and glandular part of the mucous membrane, as is shown in Fig. 18. Further, abundant bacilli, chiefly of the same short thick form previously described, can be seen in the retiform tissue of the mucosa, in the submucosa, and in the muscular coats. Fig. 19 shows the appearance of an artery and two veins in the outer muscular coat; the veins are swarming with bacilli.

In both these cases the objection may be offered that the inflammatory changes and the presence of bacilli may be the result rather than the cause of the inflammation of the peritoneal coat of the appendix. On the general evidence, however, such an objection should have little weight, and I have since examined two appendices in general peritonitis from a known cause without finding these changes.

At any rate, it is certainly worth while to examine the appendix microscopically in such cases, for it is not always possible to detect disease of its mucous membrane with the naked eye. And I am sure that in an exploratory operation in a case of general peritonitis the appendix may be examined by the surgeon and acquitted, while it is nevertheless the cause of the mischief.

A suggestive instance is to be found in a case mentioned by Pilcher.[58] A girl aged 12 was suddenly seized with the symptoms of perforation of the appendix and commencing general peritonitis. She was too ill at first for exploration, but on improvement setting in the abdomen was opened: the appendix, it is stated, was found to be healthy, but there was general purulent peritonitis, for which no cause could be found.

Peritonitis consequent upon disease of the appendix.

It seems to be probable, then, that when a general peritonitis or a local peritonitis with the formation of pus (perityphlitic abscess) arises from a diseased appendix, the actual agent which provokes the peritoneal inflammation is a microbe derived from the interior of the tube.

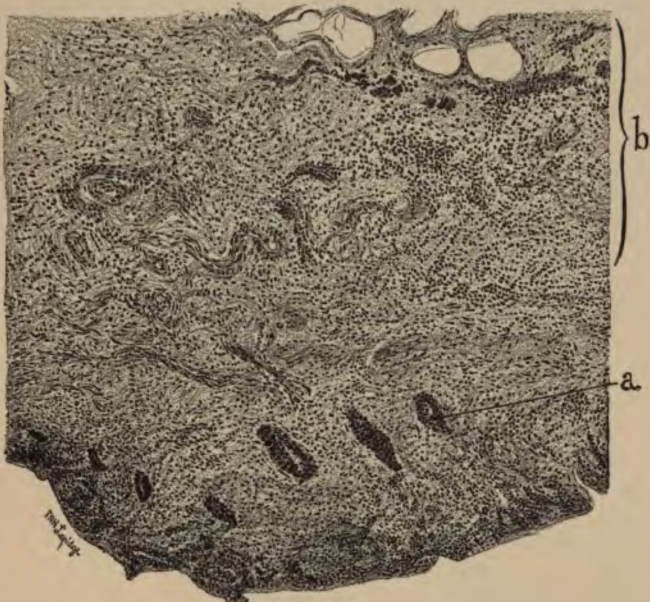

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Fig. 18.

Transverse section through the mucous membrane of an appendix, which showed no sign of disease to the naked eye. It was taken from the body of a man who had died from general peritonitis, for which no cause was found at the post-mortem examination.

a. Remains of a crypt.

b. Infiltrated submucous tissue.

(a) Bacillus coli communis.

It is possible that more than one species of bacterium may act as the exciting agent, but the evidence points to bac. coli communis as being the form which is chiefly concerned.

Hodenpyl (*loc. cit.*) has the credit of making a definite attempt to connect the presence of this bacillus with appendicular peritonitis, though some isolated observations had been made two or three years before.

In eleven cases of general peritonitis and of perityphlitic abscess (the majority being of the latter kind) he found bac. coli communis in the pus—a pure culture in ten of them, but mixed with streptococcus pyogenes in one case of local abscess which was opened on the fifth day. In this last case both bac. coli communis and streptococcus pyogenes were found in the interior of the appendix as well as in the abscess around it. In one case the perityphlitic abscess was associated with pylephlebitis and hepatic abscess, and here the bac. coli communis was obtained as a pure culture from the pus in the liver as well as from the pus around the appendix.

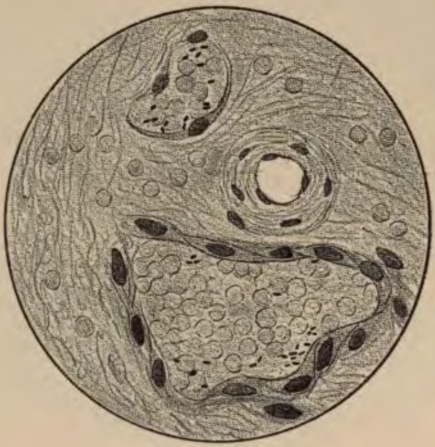

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Fig. 19.

Short bacilli in two veins in the outer muscular coat of the appendix from which Fig. 18 was taken.

He has further collected twenty-four similar cases, in all of which bac. coli communis was found in the exudate, as a pure culture in twenty-three of them, combined with streptococcus in one. These cases are reported by Welch, Robb, Clado, Adenot, Fränkel, Malvoz, Ekehorn, and Fowler.

Many other observations have been made, all telling the same tale. Roswell Park (*loc. cit.*) found a pure culture of this bacillus in a perityphlitic abscess, and again in two cases of local abscess around a gangrenous appendix. In nineteen out of twenty-three cases of appendicular peritonitis, Tavel and Lanz found it either in the appendix or in the exudate around it. In several cases it was accompanied by streptococcus, and in two cases by micrococcus pneumoniæ crouposæ. In one case the only microbe discovered was staphylococcus pyogenes citreus.

So that in sixty-one cases of general peritonitis or perityphlitic abscess consequent upon disease of the appendix, the bac. coli communis was found in the exudate in fifty-seven, and in fifty of these cases it was the only microbe present.

The almost constant presence of this bacillus in the interior of the appendix has been already established, and it has been shown by Hodenpyl and others that the healthy peritoneum in the right iliac region is practically free from bacteria.

We stand, therefore, face to face with the facts. We must either conclude that the presence of this bacillus in the purulent exudate is an accidental and subsidiary accompaniment of this form of peritonitis, or we must believe that it is the veritable cause; and if, as I conceive, the passage of bacteria through the appendix-wall may be taken as an observed fact, there can be no hesitation in deciding that the clue to the disease lies in the action of this bacillus.

(b) Other bacteria.

At the same time, it must be remembered that in some cases pyogenic cocci are found both inside the appendix and in the exudate around it. Hodenpyl obtained streptococcus in one case, Welch found it in one case, Tavel and Lanz found it in several cases, and micrococcus pneumoniæ in two cases, and in one case staphylococcus pyogenes citreus was the only form present.

So that it is possible that in some cases a pyogenic coccus may be the agent which excites the peritoneal inflammation. And although the observations as to the occurrence of bac. coli communis as a "pure culture" in peritoneal exudate are the work of the most competent bacteriologists, yet all such observations are at present open to a known source of error.

This bacillus grows in all media with such rapidity and luxuriance that a slower-growing form may be swamped and remain undetected. Barbacci (*loc. cit.*) has found that in perforative peritonitis, produced experimentally in dogs, this bacillus alone survives in the peritoneal cavity. The other microbes which pass into the serous cavity with the fæces certainly develop, but soon disappear. And he has further shown that in many cases, where cultures give only bac. coli communis, inoculation of the original exudate into an animal yielded also the micrococcus pneumoniæ.

So that, while we hold to the theory that appendicular peritonitis accompanied by the formation of pus depends on the action of bacteria which have escaped from the interior of the tube, and while we maintain that the evidence points to bac. coli communis as the usual agent, a possible similar action on the part of the pyogenic cocci must be kept in view.

It is not necessary for the present purpose to enter at length into the part played in the causation of disease by this bacillus.

The pathogenic power of bacillus coli communis.

Its reputed rôle is being continually widened, and its occurrence under various conditions is full of suggestion.

It was first described by Escherich in 1886, but Laruelle (*loc. cit.*) in 1889 was the first to show its pathogenic power, and the evidence on this point is now abundant.

As already mentioned, Hodenpyl found it in an abscess of the liver. Veillow and Jayle[59] found a pure culture of it in a dysenteric abscess of the liver. Passet in 1885 obtained an organism from a peri-rectal abscess, which he named bacillus pyogenes fœtidus, and this has since been identified with the bacillus coli communis. Muscatello[60] took a pure culture from a peri-rectal abscess, and this injected subcutaneously into guinea-pigs resulted in abscess with stinking pus, from which the bacillus was again recovered. Gilbert and Girode[61] found a pure culture in two cases of suppurative inflammation of the gall-bladder. This is a very suggestive observation, and it has frequently been confirmed. Welch (*loc. cit.*) found it in the organs of a body in a case of pancreatitis with fat-necrosis, and in this case the existence of a diphtheritic colitis may point to the mode of entrance of the bacillus into the circulation.

Marfan and Leon[62] give two cases of ulcerative colitis in which it was present in all the organs.

And in other severe disorders of the intestines it has been found after death in various organs—liver, spleen, kidney, and glands (Rossi-Doria[63] and Wyss[64]).

Laruelle has found it in the pleura in cases where pleurisy has been associated with peritonitis, and this is of great interest, since pleurisy not uncommonly makes its appearance in the course of appendicular peritonitis. Macaigne obtained it by puncture of the lung before death in a case of pneumonia associated with peritonitis. Fisher and Levy[65] speak of its presence in the lung in two cases of strangulated hernia with a pneumonic condition.

The evidence from the experimental side tends in the same direction. Small amounts of a pure culture of bac. coli communis injected into the circulation of guinea-pigs produces death in one to three days. There are found severe catarrhal lesions of intestines, swollen Peyer's patches, and unusually intense congestion of the peritoneum with bloody fluid in its cavity; and the bacillus can be recovered from the blood and solid viscera (Escherich).

As regards the results when it is introduced into the peritoneal cavity, the varying virulence of the bacillus has been already alluded to as being an important factor in the etiology of peritonitis.

A pure culture from a healthy bowel seems to have no harmful effect upon the peritoneum. It becomes virulent, however, when taken from a bowel which is the seat of such conditions as inflammation, strangulation, or œdema. When diarrhœa is present, it assumes virulence, and possibly also in constipation.

When taken from a case of suppurative peritonitis, it invariably reproduces the same condition if injected into the abdominal cavity of animals. Ekehorn's observation on this point has been already quoted.

Finally, there is evidence tending to show that its virulence, when introduced into the peritoneum, is dependent to some extent on the fluid with which it is mixed. And this is an important point when we consider the wide variation in the clinical forms of appendicular peritonitis. Laruelle (*loc. cit.*) has shown that its activity is much increased if sterilised fluid from the intestine is injected with it. When injected with salt solution the result is negative, but mixed with sterilised water or ox-gall a rapidly fatal peritonitis ensues, neither the sterilised water nor the ox-gall giving this result alone. Blood also seems to be a very favourable medium. All this is quite on a level with Cheyne's experiment on the action of proteus vulgaris, where the virulence of the organism was shown to depend on the medium with which it was mixed.

(B) PERITONITIS ACCOMPANIED BY A COAGULABLE EXUDATION, WHICH IS CAPABLE OF UNDERGOING ABSORPTION OR ORGANISATION (PERITYPHLITIS)

While I believe that the bacterial origin of an acute general peritonitis and of a perityphlitic abscess is established by the facts detailed above, the origin of the local adhesive peritonitis manifested by a coagulable exudation around a diseased appendix still remains in doubt. This is by far the most common form of the disease.

It may be said that these cases are never fatal except from some complication or accident, so that no information as to the bacteriology of the exudate can be obtained from the post-mortem room. In this country they are seldom subjected to operation, and in American records of cases, where the appendix is excised at an early stage, I can find no observation as to the presence or absence of bacteria in the coagulated exudation.

We have to consider on the one hand the existence of chronic inflammation of the mucous membrane of the appendix continued through many months or many years, and on the other hand the occurrence, from time to time in this period, of brief attacks of inflammation of its peritoneal coat and of the adjacent peritoneum.

Are we to believe that in each such attack the process which we call "inflammation" spreads from the mucous membrane to the submucosa, and from the submucosa to the muscular coats, and thence to the peritoneum? I think one may well be sceptical as to such an extension of inflammation from the mucous to the serous coat either in the appendix or in any other part of the intestine. Fifty years ago it was thought to be a common event.

And I think it is not improbable that subsequent observations may lead us to look upon all forms of appendicular peritonitis as being alike in owing their causation to bacterial infection. It is true that the clinical difference between the various forms of peritonitis is very great. It may entail a week's illness, in which the patient has scarcely any constitutional disturbance, and is with difficulty kept to his bed; or in forty-eight hours it may put him beyond help from man. And yet between these two extremes there is a perfect unbroken gradation.

We have classified the forms of appendicular peritonitis according as they are general or local, suppurative or adhesive. This division is useful, but it is rough, and not true. There is no such absolute distinction in the natural history of the disease. As regards the extent of peritoneum inflamed, there is every degree between an absolutely general peritonitis and one involving the surface of the appendix only, and as regards the character of the exudation there is every degree between an abdomen full of pus and a trace of coagulated fibrin on the floor of the right iliac fossa.

If, as is probable, all these forms should be regarded as having a similar origin, we may perhaps partly explain their wide differences by variation in the virulence of the micro-organism concerned. But I believe that the explanation of the clinical differences will be found to lie chiefly in the manner by which the infection arises, and consequently in the surroundings of the micro-organism at the moment of infection. In one great class of cases it gains entrance into the peritoneal cavity in company with fæcal matter or with the products of necrosis of the appendix-wall; in the other class the action of the micro-organism is unaided by any such material.

In the absence of direct evidence, however, nothing certain can be said on this point.

CHAPTER VII

Classification of the Forms of Appendicular Peritonitis.

Simple Perityphlitis.—Exciting causes, onset of symptoms, course of illness, duration, result.

Perityphlitic Abscess.—Exciting causes, previous attacks, onset of symptoms, course, diagnosis of formation of pus, sources of error, result, position of abscess, extra-peritoneal suppuration, lumbar abscess, abdominal abscess. Complications and sequelæ, intestinal obstruction, pylephlebitis, pyæmia, fæcal fistula, inflammation of muscle, œdema, hæmorrhage, pleurisy, appendicitis in hernial sac, perityphlitis and pregnancy. Appendicular colic, actinomycotic abscess, pelvic peritonitis in women, carcinoma of cæcum.

General Peritonitis.—Onset, symptoms. Cases with gradual onset, cases with sudden collapse. Treatment adopted. Complications.

Relapsing Perityphlitis.—Frequency of recurrence, condition of appendix, symptoms, result.

CLINICAL DESCRIPTION OF THE FORMS OF PERITONITIS ARISING FROM DISEASE OF THE APPENDIX

Classification of the forms of appendicular peritonitis.

RECENT systematic writers have shown a tendency to subdivide the various forms of appendicular peritonitis somewhat minutely, losing thereby in clearness what they gain in theoretical accuracy. From a practical point of view the classification already adopted would seem to be sufficient. There are three chief forms to be recognised—viz.:—

(1) An adhesive peritonitis limited to the right iliac fossa, accompanied by the exudation of a coagulable material (perityphlitis).

(2) A similarly localised inflammation, proceeding to the formation of pus (perityphlitic abscess).

(3) A general peritonitis.

Each of these three forms of peritonitis follows upon some one definite morbid condition of the appendix, as already described, so that in such a classification pathological observation and clinical experience are brought into line.

But it must be confessed that there are certain cases as to which our clinical experience has outrun our knowledge of pathology. There are cases which in one or other important respect take an unusual course, and we are often unable to explain this unusual course by any unusual feature in the morbid conditions actually observed. There are, for example, (i.) cases in which a perityphlitic abscess may be formed and be localised by adhesions, and yet a sudden general peritonitis may supervene. (ii.) In other cases, again, a general peritonitis of sudden origin may slowly subside, leaving a recognisable inflammatory mass in the right iliac region, such as is characteristic of a simple perityphlitis. (iii.) There are also cases, happily rare, which are characterised by their deceptive nature, where grave destructive lesions of the appendix are attended with but little constitutional disturbance, and all previous experience is hopelessly at fault. (iv.) In many cases, again, the patient passes from one attack into another with little or no interval. All these clinical varieties require some special mention.

(1) LOCAL ADHESIVE PERITONITIS (SIMPLE PERITYPHLITIS)

Apparent exciting causes.

The evidence already put forward points to the common origin of this form of peritonitis in a chronic catarrhal inflammation or in a cystic condition of the appendix, though in some few cases a concretion is the cause.

In any case the disease of the appendix is probably in existence for some length of time before the peritoneal inflammation is excited, and, as might be expected, in a certain number of cases an apparent exciting cause can be traced.

In some cases an indigestible meal seems definitely to usher in the symptoms, though it is open to any one to consider that this is nothing more than a coincidence. Such a meal acts probably by causing strong peristaltic movements either of the small intestine or of the cæcum, and so shifting the relations of the diseased appendix. Possibly some pressure may be thus exercised upon it, and possibly the appendix itself may show the same unwonted contractile movements as do other parts of the intestine upon the receipt of indigestible material.

In twelve of our 190 cases of simple perityphlitis such a history was obtained. In most of the instances the symptoms, at first attributable to the ingestion of the irritant, passed in the course of six to twelve hours into the definite symptoms of perityphlitis. A man, perceiving on his chest a syphilitic eruption, took a dose of brimstone and treacle; purging and vomiting ensued, and the next day pain in the right iliac region occurred, followed by the appearance of a definite inflammatory mass in this position. This case proved to be of the relapsing form, and the patient underwent two subsequent attacks in the course of the year. A girl indulged freely at a school-treat, and on her return in the evening was seized with diarrhœa and vomiting; on the next day began an attack of perityphlitis.

One man ate a quart of mussels twelve hours before his attack commenced. A boy ate a large quantity of sardines in the middle of the day; in the course of the afternoon he was very sick, and in the evening was seized with severe pain over the whole abdomen; for the next two days he remained exceedingly ill, and on the third day the pain shifted to the right iliac fossa, where an inflammatory mass could soon be felt.

In other cases some definite violence is offered to the district of the appendix. In sixteen instances out of 190 was this the case. A militia-man fell from a horizontal bar upon his belly, and therefrom ensued the usual phenomena of perityphlitis. A young man helping to carry a slab of slate let his end fall, and in trying to save it he slipped and strained his right side: shortly afterwards he was sick and was seized with pain around the appendix, followed by the usual signs and symptoms of a local peritonitis. A strain while throwing gravel, while rolling a barrel, while lifting a heavy piece of furniture, a blow in the right groin from a fist, a kick, a thrust from the handle of a mop, and a dig from the handle of a spade were all followed in the course of a few hours by unmistakable signs of perityphlitis. In one similar case suppuration occurred, and a calcified concretion was found in the pus.

In the majority of instances, however, no such immediate cause can be detected. The symptoms may appear in any individual at any moment.

The onset of symptoms

The first symptom is almost invariably pain in the abdomen. This pain is of sudden onset, and generally of some severity. In about half our cases it was almost from the first localised in the right iliac region, but in at least half the cases it was at first felt either all over the abdomen or in some part of the abdomen other than the neighbourhood of the appendix. And yet when this is the case there is as a rule no reason to suspect an inflammation of any other part of the peritoneum than that surrounding the appendix. For the pain shifts to the right iliac fossa in the course of twenty-four or thirty-six hours, and remains localised in that position throughout the illness. In a great many cases the initial pain is felt about the umbilicus or across the abdomen below that point.

The pain may be dull and aching; sometimes the patient uses such words as "cramping" or "griping" to describe it. It may be slight, and a man will try to walk it off. But it may be very acute: a child may suddenly cry aloud at its onset, and even a man may sweat and grow faint. At times it is so sharp as to simulate a renal or biliary colic.

In many instances the patient may vomit once or twice within a few minutes or hours of the onset of the pain. But the vomiting is seldom an urgent symptom. As a general rule it may be said that the more severe the vomiting the greater is the extent of the peritoneum involved. This is a truth when a large number of cases are investigated, but it gives little help when examining an individual case, for the vomiting of this local peritonitis may be violent and continued, while in a general peritonitis that is likely to prove fatal it may on occasion be trifling. In relapsing cases it may be noted that the vomiting is often less severe in the later than in the earlier attacks.

Probably the temperature rises to some extent from the very first, but hospital experience does not allow of observation on this point. In one case, that of a child, admitted into the hospital for an alveolar abscess, I was able to watch the actual development of the symptoms of a typical mild attack. Within two hours of the onset of the pain the temperature was 104·6, which was the highest point reached in the course of the illness (see Chart 1).

In nearly all cases, unless a purgative is given at the commencement of the attack, constipation sets in from the first, but in a small proportion the initial pain and vomiting are accompanied by two or three actions of the bowels. This occurred in eight of our 190 cases. This again, like the vomiting, is rather more apt to occur with a general than with a local peritonitis.

In some instances the onset of the attack is accompanied by a general feeling of illness as well as by the more particular symptoms above described. There may be headache, a feeling of coldness (seldom or never an actual rigor), and a sensation of weakness. Occasionally a veritable condition of collapse may accompany the onset of a simple attack of perityphlitis which is not otherwise severe.

The course of the illness. (1) *Pain.*

The pain is at its worst in the first two or three days of the illness, and by the time the patient comes under treatment it has almost invariably become localised in the right iliac region. In most instances the patient is quickly brought more or less under the influence of opium, but apart from treatment, and even in cases where purgatives are given, the localised pain is rarely felt after the first four or five days, so long as the patient remains at rest.

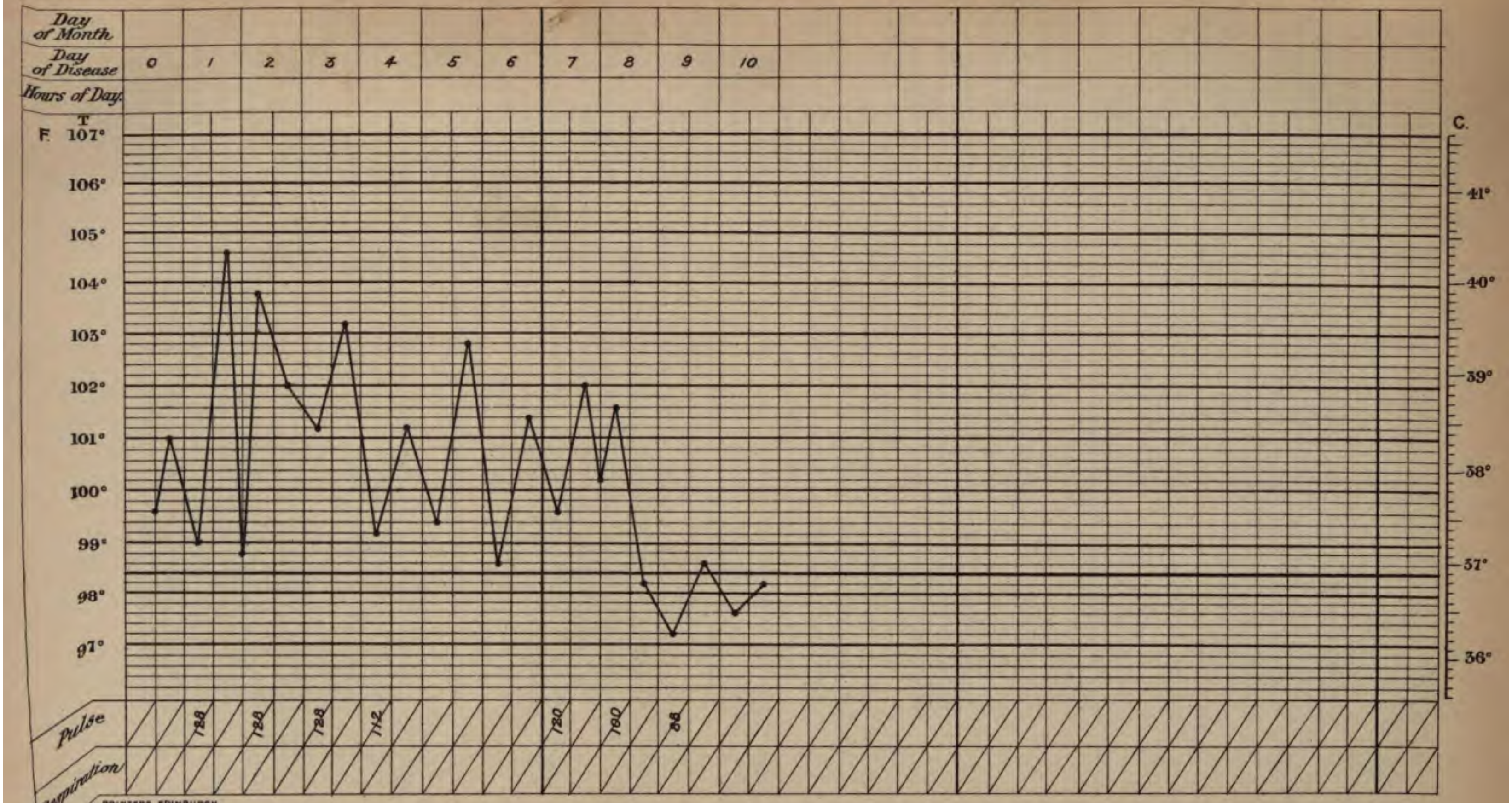
In some few cases (five in our 190) some pain attends the act of micturition, which is probably to be explained by a pelvic position of the appendix and consequent involvement of the peritoneal surface of the bladder.

Occasionally pain is complained of down the front of the thigh, in the distribution of the branches of the anterior crural nerve.

(2) *Tenderness.*

Tenderness in the right iliac region is a constant feature, as might be expected, and this persists after the acute pain has ceased. The amount of tenderness present may be taken as a fair indication as to the progress towards recovery.

Name C.B. *Age* 14 *Disease,* Simple Perityphlitis *Index No.* 1.



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In 1889 McBurney[76] drew attention to a certain spot, between 1½ and 2 inches from the anterior superior spine of the right ilium along a line drawn from that process to the umbilicus, at which deep pressure always elicits pain, when the appendix is diseased. This spot marks roughly the base of the appendix when the cæcum occupies its usual position. He claims distinct diagnostic value for this tender point.

Its existence and value have been investigated in all quarters, and the published accounts are conflicting.

The experience derived from our cases goes to show (1) that it is present in all cases of local peritonitis arising from a diseased appendix; (2) that it is not marked in cases of general appendicular peritonitis, and so it fails us when it is most required; (3) that it is certainly present in some other abdominal conditions, such as pelvic peritonitis in females.

I am inclined to think that its chief use lies in the early period of a local peritonitis, at the time when the disastrous purgative is so commonly administered. But hospital practice affords very little opportunity for testing it at this stage.

(3) *Appearance.*

In severe cases the patient presents some degree of the sunken-eyed anxious appearance so characteristic of general peritonitis, but as a rule the face expresses nothing more than the early pain, or it may be is somewhat flushed by the fever.

(4) *Digestion.*

The tongue is always more or less furred, but seldom shows any tendency to become dry. The appetite is lost, but the distressing thirst of general peritonitis is not felt. Vomiting is very rarely met with after the second day of the illness.

(5) *Condition of the bowels.*

It has been already said that the onset of the illness may be marked by a tendency to looseness of the bowels. In a few cases (6 in our 190), which are in all other respects of the ordinary type, more or less diarrhœa is present throughout, but with these exceptions constipation is by the nature of the disease absolute in its whole course. Even after the cessation of the more acute symptoms, there may be difficulty in procuring an evacuation of the bowels by enema. In some cases the first stool after this period of constipation contains mucus and even a trace of blood.

(6) *Urine.*

The urine is generally such as is found in other febrile conditions. It often contains indican. In only one of our cases did it contain a trace of albumen attributable to the high fever.

(7) *Temperature.*

The character of the temperature-rise is an important consideration. If we take it that there is a rise of temperature from the first onset of the abdominal pain, as is most probable, it appears from our cases that the common duration of the fever is roughly from six to ten days. As a rule there is a steady fall in the temperature from the first day until the last, so that a chart has a very regular and constant appearance.

The initial rise of temperature commonly reaches 102 to 104. In some cases 105 is reached (Charts 1 and 2).

If the fever were always of this character, the matter would be simple. But, as will be shown later, the recognition of the presence of pus in the region of the appendix depends in part upon the behaviour of the temperature, and some occasional variations in what may be called the normal temperature-chart of perityphlitis are misleading in this respect. In some instances a steady rise of temperature sets in during the course of the illness, strongly suggestive of the formation of pus, and yet the illness may be as uneventful as usual (Chart 3). Indeed, the maximum point may be reached just before the peaceful end of the illness in resolution (Chart 4).

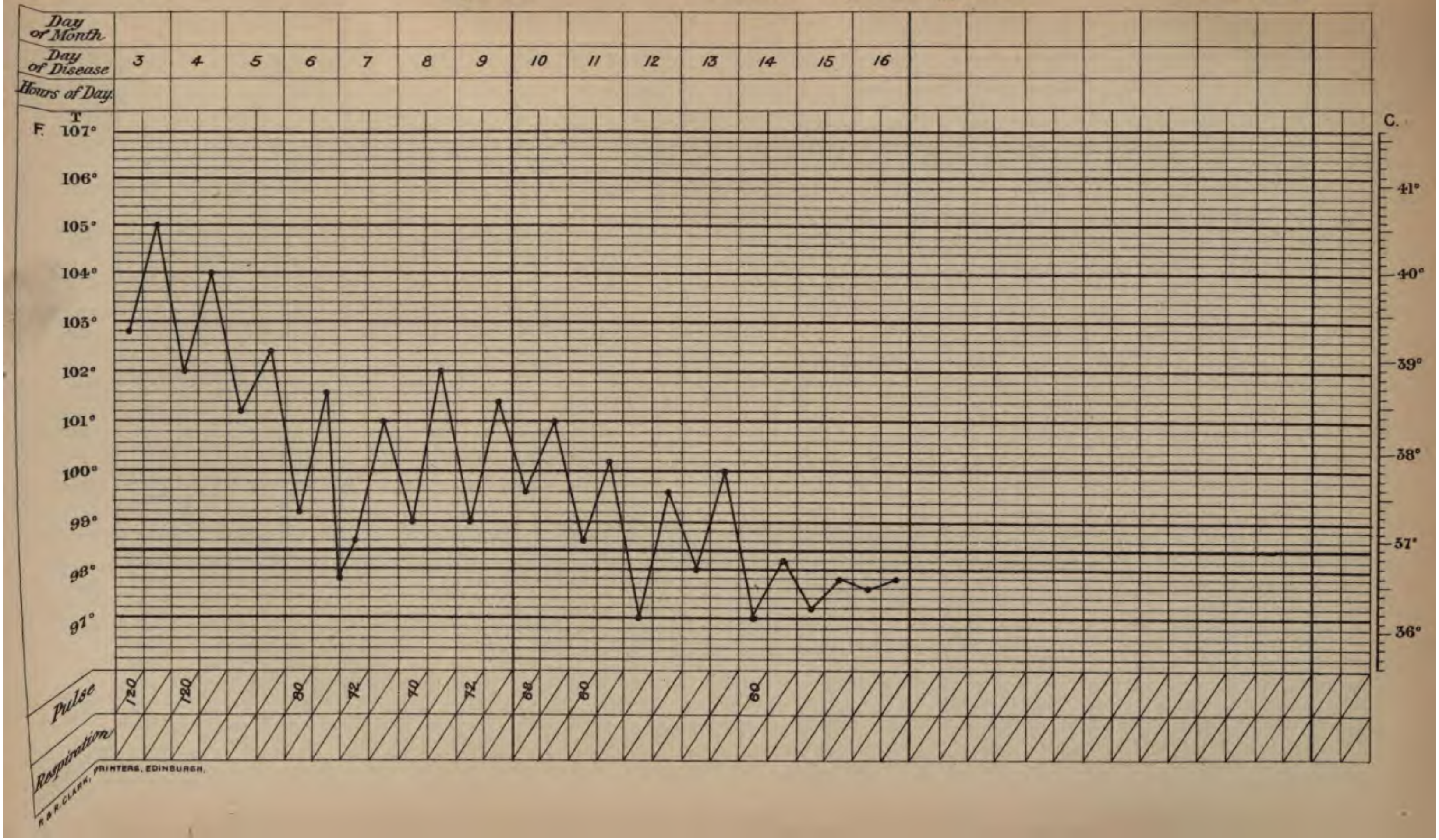
In these last two charts a not uncommon phenomenon is seen—viz. an abrupt cessation of the fever, which is often as sudden and complete as in pneumonia. At least ten instances of this could be drawn from our 190 cases. The subjoined chart is a good example of this (Chart 5).

In the case from which Chart 4 was taken, a very large inflammatory mass was still present in the right iliac fossa, and the temperature was still over 103 on the ninth day of illness. I was confident as to the presence of pus, and arranged for its evacuation on the following day, but in the next twelve hours the temperature fell to normal, and the patient rapidly recovered without assistance. In some of these cases of a sudden fall in a high temperature it is highly probable that it coincides with the discharge of a small abscess into the bowel, and instances of this will be given later. But in the cases already quoted no evidence of this could be found in the subsequent stool. In one case, immediately upon the drop in the temperature I cleared out the large bowel as well as possible by an enema, but could find no trace of pus or blood.

Occasionally the period of fever is much prolonged. In these cases the patient has usually had one or more previous attacks, and it is probable that the long duration of the fever is due to a minute collection of pus lying deeply among the adhesions. The fever may thus continue, with perhaps a day or two of normal temperature, over three or four weeks, as in Chart 6, without anything more tangible than tenderness and resistance in the region of the appendix. In some of these cases of prolonged fever the descent of the temperature is steady and gradual throughout, as in Chart 7, and some are probably mistaken for typhoid fever.

Further, a study of the temperature makes it clear that the height of the fever has no constant relation to the size and extent of the inflammatory mass in the groin. High fever may subside rapidly or suddenly, while the inflammatory hardening may remain unchanged or slowly follow suit.

Name H. H.— *Age* 17 *Disease* Simple Perityphlitis *Index No.* 2.



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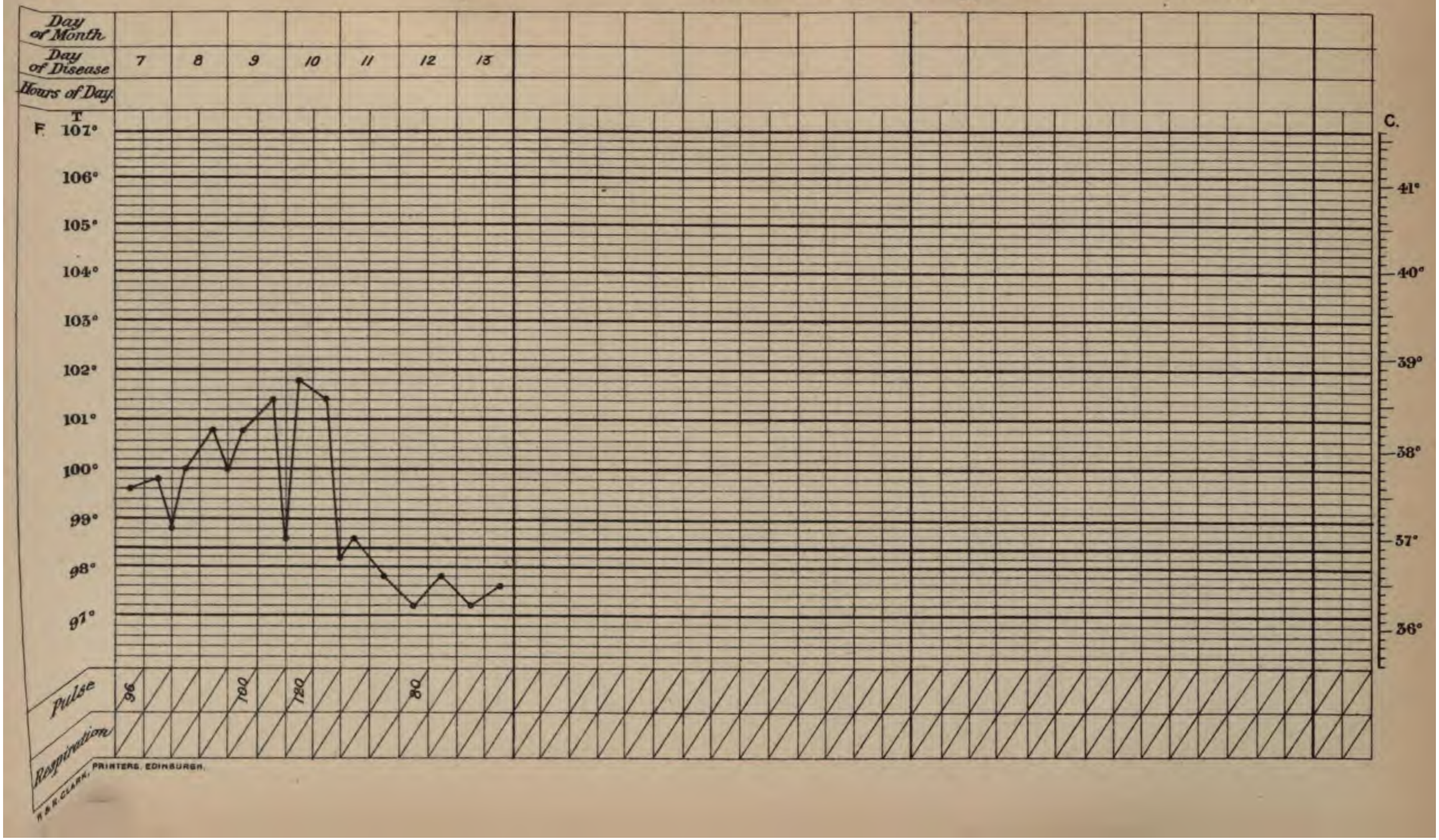
Name A. G. – *Age* 15 *Disease* Simple Perityphlitis *Index* No. 3.



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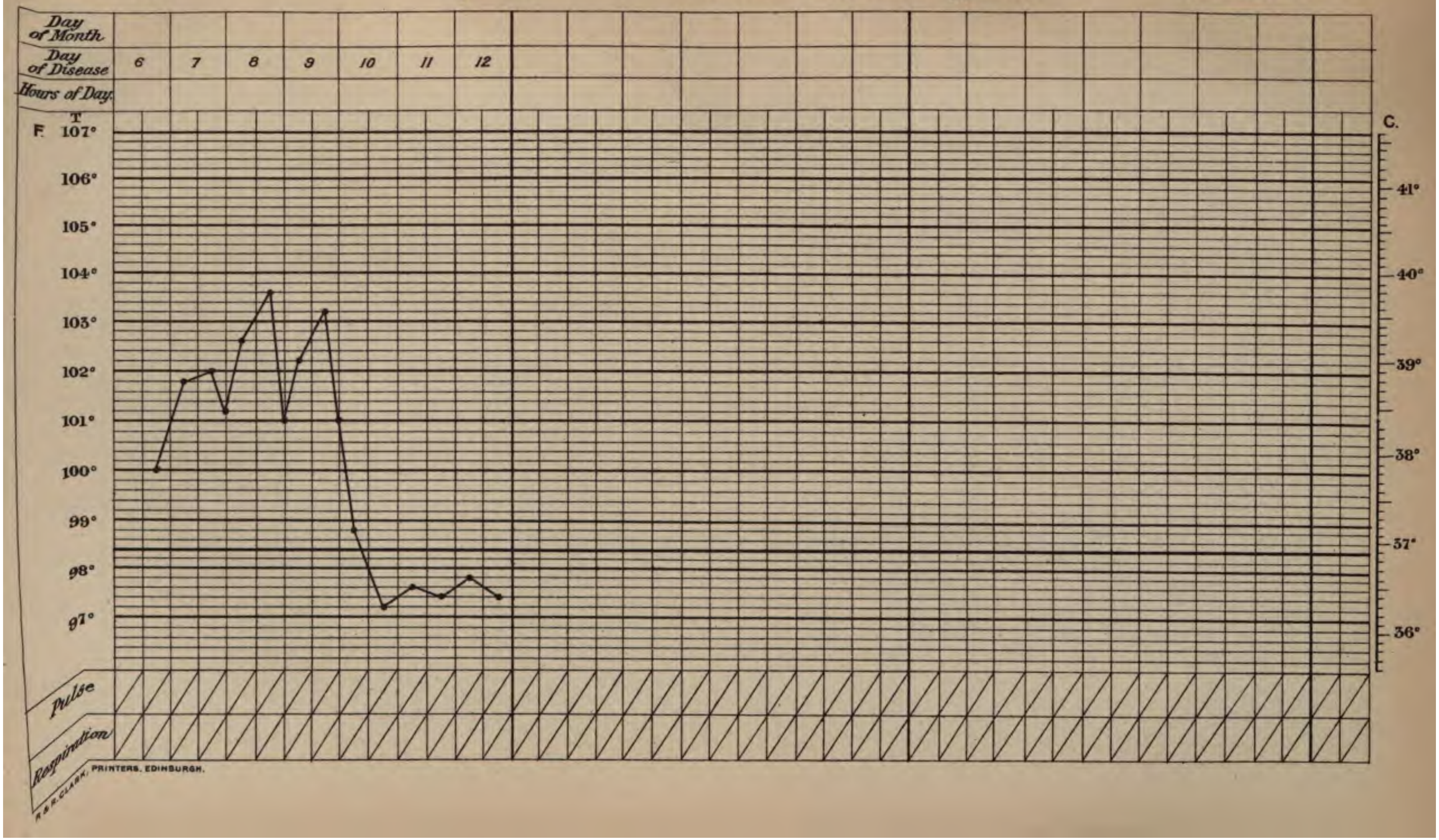
Name *G.P.* Age *10* Disease *Simple Perityphlitis* Index No. *4.*



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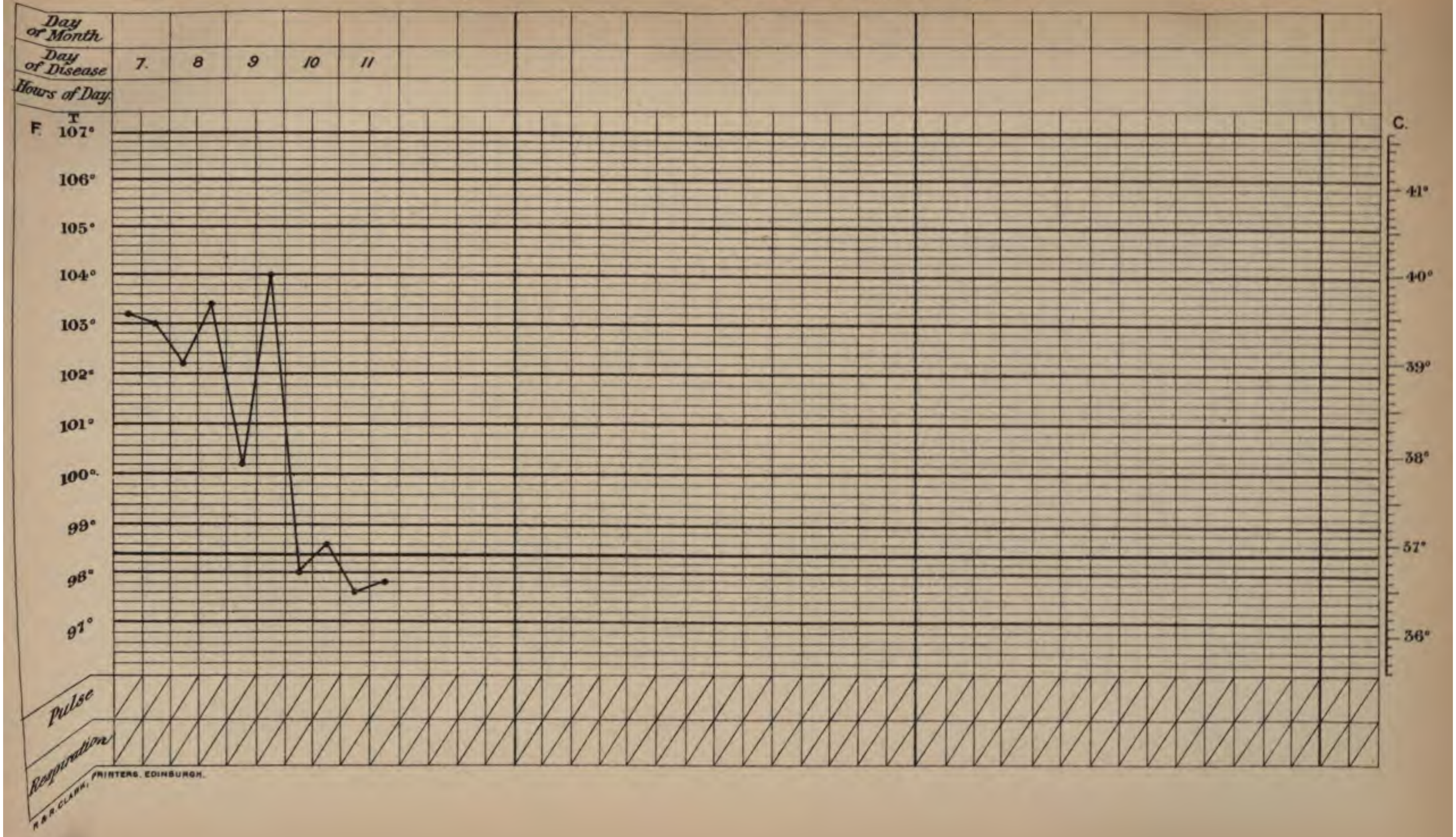
Name C.B.— Age 17 Disease Simple Perityphlitis Index Nº 5.



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(8) *Pulse.*

The pulse is quickened in proportion to the fever, but is seldom affected in point of strength or tension.

(9) *Physical signs in the abdomen.*

Almost from the commencement of the illness, as already stated, there is some degree of tenderness in the right iliac region. In fact, in an early stage tenderness here may be the only guide to a diagnosis of the condition. It is sometimes very acute, and it is never absent. It may be experienced only in a small area, but its extent necessarily varies with the amount of peritoneal surface inflamed. There may be acute tenderness over the whole of the right lower quadrant of the abdomen: its area may extend up to the right costal margin, and also round into the flank. In many cases at an early stage there is also a purely cutaneous hyperæsthesia over a similar extent.

As a rule, when patients are seen in the first two or three days of the illness, the abdomen is found to be slightly distended, and over its right side there is a rigid contraction of the muscles. This muscular rigidity may continue as long as any acute symptoms are present, so that nothing can be said of the right iliac region except that it is more resistant th n other parts of the abdomen. As a rule, however, this rigidity soon passes off, and something of the nature of a tumour can then nearly always be felt in the iliac fossa. There may be a soft but resistant mass here, suggestive of thickened intestine, and this may have no definite margin in any direction. Generally, however, the mass is fairly hard, and its upper and inner limits are to be plainly felt. It varies greatly in size. It is commonly somewhat oval in shape, with its long axis lying above and parallel to the outer part of Poupart's ligament, and it has then about the size of a hen's egg. It may, however, extend inwards to the middle line of the abdomen, and upwards to the level of the umbilicus, thus assuming a quadrilateral shape. It may extend round into the flank, producing a sensation of hardening around the ascending colon. It is sometimes triangular in shape, with its base situated upon Poupart's ligament and its apex pointed towards the umbilicus. Occasionally it is situated nearer the middle line than usual, so that its centre lies vertically over the true brim of the pelvis. The inflammatory mass may in an adult measure as much as four inches in transverse and vertical lines, and yet it may rapidly subside without any evidence of the formation of pus.

I have never in any case of simple non-suppurative perityphlitis been able to feel anything abnormal per rectum, though it is said that the iliac mass can be reached in this way.

The nature of the inflammatory hardening has been made quite clear by exploratory operations. There is found in such cases congestion, œdema, and swelling of the wall of the cæcum and of the small bowel occupying the iliac fossa. The interstices between the intestinal coils contain a variable amount of coagulated fibrinous exudation. The cæcum and adjacent small bowel are thereby fixed, and it is this fixation which chiefly gives rise to the sensation of hardness.

Doubtless in many cases fæcal matter accumulates in the cæcum and affected part of the small bowel, and so adds to the size and hardness of the mass, but this must be considered as a minor element.

In some cases also thickening of the omentum in this region takes part in the formation of the mass.

Resonance on percussion is generally impaired over the surface of these inflammatory tumours, especially when they reach a large size. But it is possible to feel a deep-seated tumour very distinctly, and yet to find no alteration in the note over it. In this case the inflammatory exudation is probably small in amount and deeply situated in the iliac fossa, with comparatively healthy small intestine lying over it. In fact, the note may sometimes be tympanitic, indicating a tensely distended condition of the superjacent bowel.

Finally, it is of great importance to realise that a patient may present all the symptoms of perityphlitis, including the characteristic pain and fever, and the illness may run the usual course, and yet there may be an entire absence of any palpable hardness in the right groin from first to last. There will, however, always be a more or less acute tenderness in this region, and on this tenderness in conjunction with the general symptoms a certain diagnosis may be based in the male. In the female the uterine appendages will always in such cases afford room for doubt.

This absence of the usual inflammatory mass is probably to be explained by an unusual position of the appendix, which is either pelvic or retro-cæcal. In one patient, whom I saw in two separate attacks, the diagnosis had in each attack to be made in the absence of tangible hardness in the iliac fossa, though there was acute tenderness at McBurney's point; at the subsequent excision of the appendix it was found to be buried in the ileo-cæcal fossa, and partly concealed by the cæcum.

The constipation which is almost a constant feature is doubtless due to a paralysed condition of the affected portion of the bowel. In some instances there is a definite indication of a forcible peristalsis on the proximal side of the paralysed part. Rumbling and gurgling are felt both by patient and physician, and for a few seconds a portion of the bowel may be seen to rise up and form a tense prominence of the abdominal wall. Especially is this the case where the tumour is a small one, and where consequently only a small section of the bowel is paralysed. It is common also for this to occur during resolution of the inflammatory mass, when a portion of the bowel may be supposed to regain its contractile power while still some portion is paralysed.

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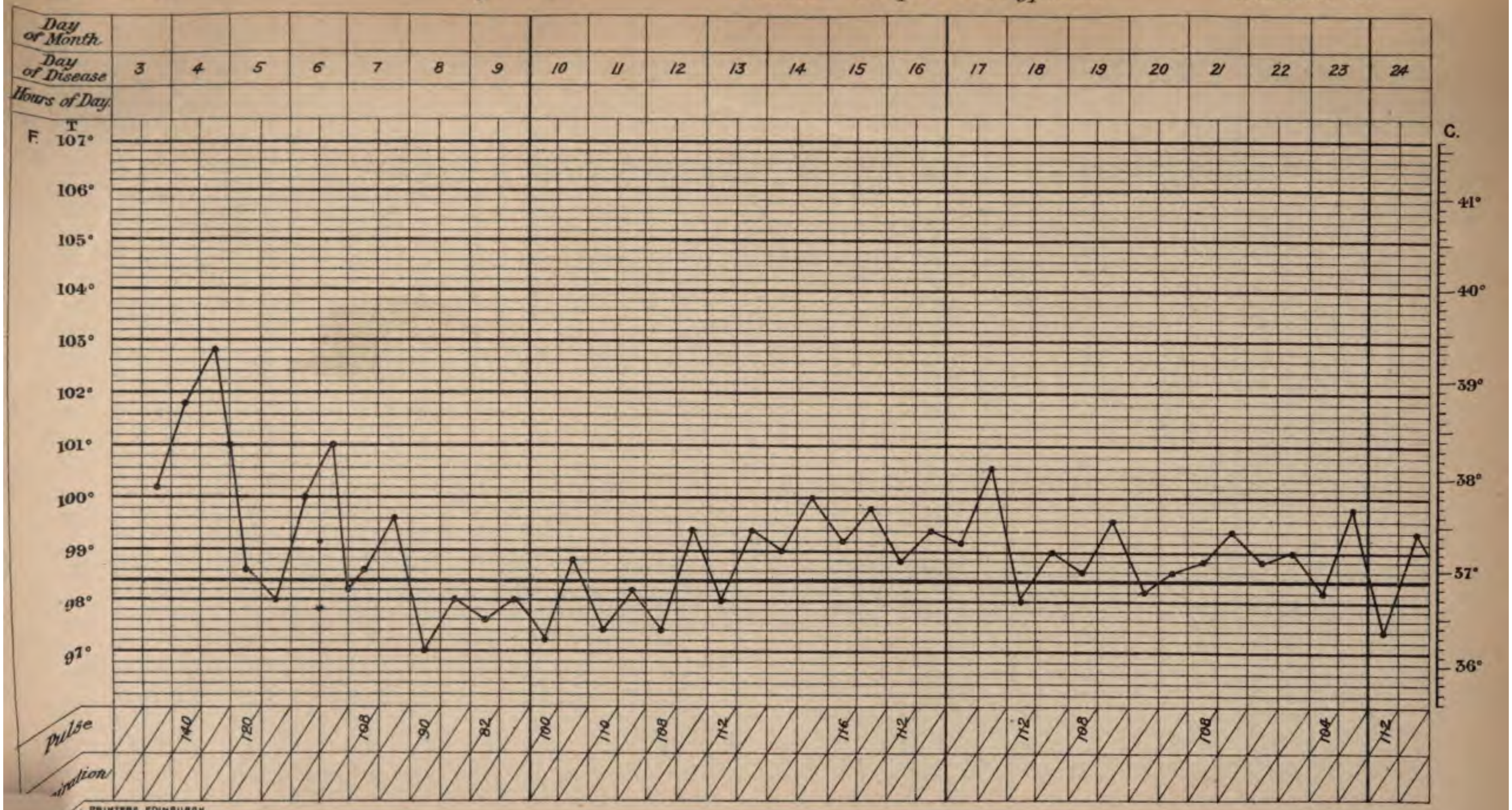

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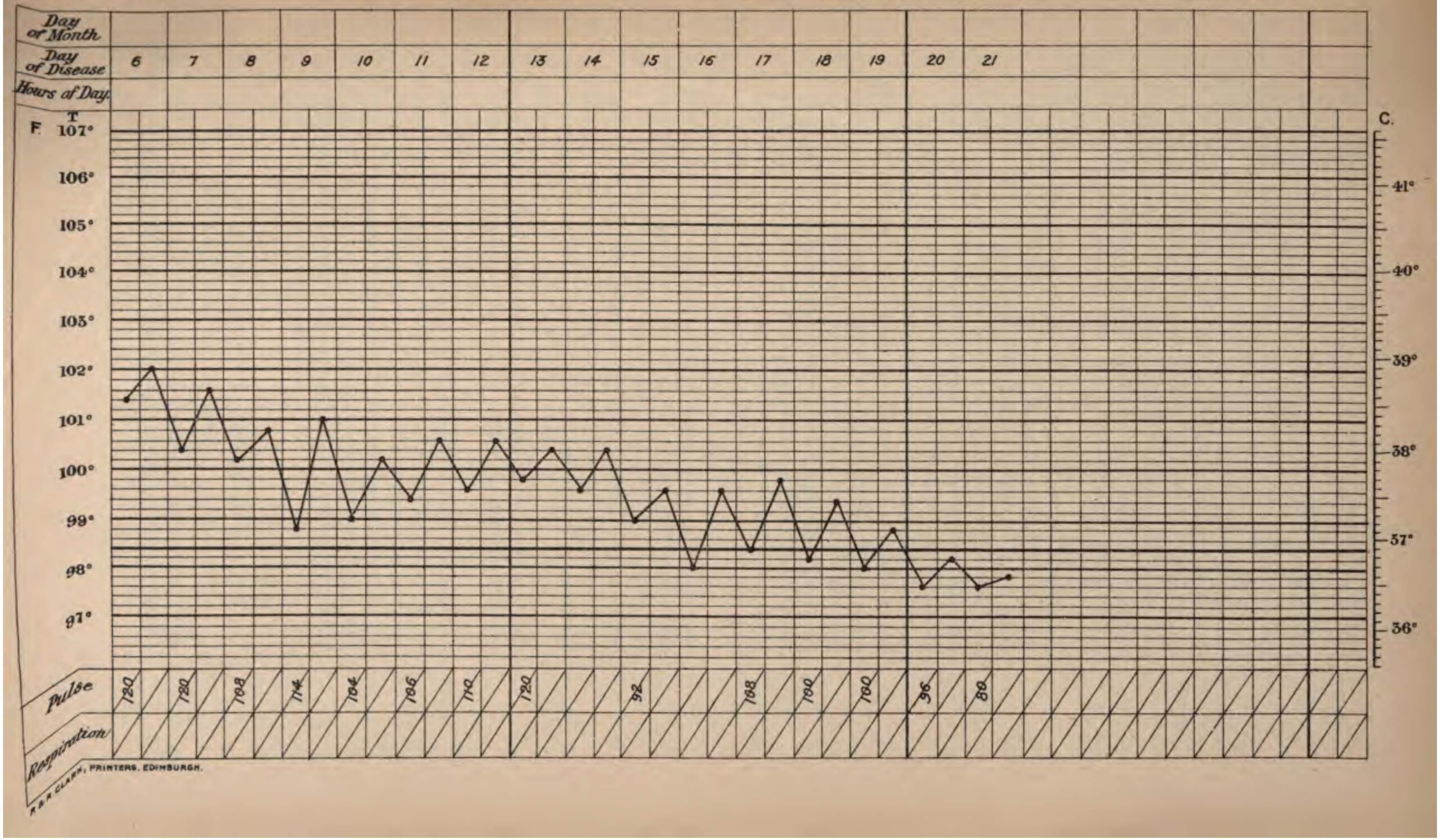
Name S. M.- Age 18. Disease Simple Perityphlitis Index No. 7.



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This irregular peristalsis is often accompanied by a paroxysmal pain, and owing to the formation of adhesions such pain and discomfort may persist when the patient is well in all other respects.

Duration of the illness.

Commonly, about the end of the first week the fever subsides, the tongue becomes cleaner, and the appetite begins to return. The inflammatory induration in the groin shows a daily decrease in size, and it is no longer tender. During the absorption of the coagulated exudation a characteristic crepitant sensation is often realised by the hand on making pressure in the iliac fossa.

It may be taken as a general rule that all acute symptoms are at an end in ten to fourteen days. Our hospital figures show on an average that it is necessary to keep a patient from work for a total period of about twenty-five days. The extremes are, however, wide apart. The patient may be free from pain and fever in four or five days or even less, the inflammatory mass in the groin may melt away in two or three days more, and he may be discharged from the hospital in ten days from the beginning of the illness. On the other hand, as has already been mentioned, the fever may continue in an irregular manner for four complete weeks, during the whole of which period there may be more or less pain, tenderness, and induration in the iliac region, with a furred tongue and constipation and some coincident wasting; and in such cases forty to fifty days or more may elapse before active life may be safely resumed.

In many cases, after the subsidence of the main inflammatory tumour, an elongated bar from one to two inches long may be felt for two or three weeks in the iliac fossa, usually lying parallel with Poupart's ligament. This is probably often the swollen appendix itself, but in some cases it has been demonstrated by exploratory operation to be part of the thickened free edge of the omentum.

I have thought that patients who present this body are particularly liable to recurrence, but the evidence is insufficient for a definite conclusion.

Result.

Of 190 consecutive cases having the clinical characters above described, all made a good recovery. The total amount of surgery employed to bring about these 190 recoveries is comprised in one successful removal of the appendix during an attack of perityphlitis. In two cases a similar attempt was made after an attack, but the strong adhesions present rendered it inadvisable to complete the excision.

(2) LOCAL PERITONITIS WITH THE FORMATION OF PUS (PERITYPHLITIC ABSCESS)

It has been shown that the conditions of the appendix, with which a local suppurative peritonitis (or perityphlitic abscess) is associated, are mainly (1) ulceration due to the presence of a concretion, often culminating in necrosis of the appendix-wall, and (2) an acute necrosis of the wall consequent upon a chronic catarrh. Less commonly a simple chronic catarrh without necrosis or a cystic appendix may prove sufficient to excite this local formation of pus.

Apparent exciting cause.

As in the case of the local non-suppurative disease, the symptoms, in the case in which pus is eventually formed, may have an apparent starting-point in an indigestible meal, a direct injury, or a violent muscular effort. In two cases out of our thirty-eight the trouble excited by an unwise meal passed into a perityphlitis in the course of twenty-four hours. In four other cases there was evidence of injury or strain. In one case a boy was knocked down, vomited in the next few hours, and in twelve hours began to show the signs of a local peritonitis in the right iliac region. A man was carrying a heavy piece of goods, when he was seized with sudden pain in the region of the appendix. A boy was attacked while drawing a heavy truck. A woman had a slight attack of the non-suppurative form, and returned to her work, which was that of a machinist, and necessitated the use of a treadle by the right foot. After a few weeks of work of this kind a local peritonitis recurred, which quickly resulted in the formation of pus.

It does not appear from the figures here given that such apparent exciting causes are to be traced more commonly in the suppurative than in the non-suppurative form, but a larger experience is required to settle this point.

Previous attacks.

In eight of these thirty-eight cases there was a history of one, two, or three previous attacks of non-suppurative perityphlitis. In three of these eight cases a concretion was found in the pus.

In one of the thirty-eight the patient had a large abscess, which was opened and drained in the usual way, and with the usual perfect recovery. After an interval of three years of good health, he returned with a second small collection of pus in the same situation. This was treated in the same way, and he has remained free from any further trouble for two years. This is the only instance which I have met with where a second perityphlitic abscess has occurred.

Onset of symptoms.

It is clear from a study of a large number of cases that as regards the onset of symptoms there is no material point of difference between the suppurative and the non-suppurative form of local appendicular peritonitis. It is important to recognise this. Whether the onset is sudden and severe or gradual and mild, no man can foretell in the first forty-eight hours whether resolution or suppuration will be the outcome. This must be realised and confessed. Neither the height of the initial fever nor the severity of the pain, vomiting, or general constitutional disturbance can be taken as giving any clue to the subsequent course of the disease.

This identity of the manner of the onset in the two cases is strongly suggestive of a common origin and a common nature, at any rate in the early part of its course. And, further, it may be said with truth that the symptoms and course of the illness in a case of perityphlitic abscess differ not a whit from those of the non-suppurative perityphlitis, until pus in some appreciable quantity has been formed.

In fact, considering the much greater frequency of the non-suppurative form, the occurrence of suppuration might almost be regarded as a complication of the simple form, and not as a separate type of disease.

Symptoms and course.

The pain and tenderness which are present from the first are not more severe than in simple perityphlitis. There is, however, rather more tendency to a looseness of the bowels, six of our thirty-eight cases suffering either from diarrhœa throughout the illness, or from alternate attacks of diarrhœa and constipation.

The tongue is furred, the pulse is quickened, and the temperature as a rule shows the same initial rise followed by a gradual decline.

The shivering, rigors, and sweating so common in connection with pent-up pus are here seldom if ever met with.

In fact, up to a certain point there is no feature in the symptoms which may enable us to distinguish between a suppurative and a non-suppurative case, and the physical signs in the abdomen are identical. It is consequently probable that up to this same point the two cases are of the same nature.

Instead therefore of recapitulating the signs and symptoms, it will be sufficient to indicate the grounds on which an early diagnosis of the presence of pus may be made.

Diagnosis of the formation of pus.

I am confident that in a great majority of these cases of perityphlitic abscess we must rest our diagnosis of the presence of pus entirely upon a "continuance or increase of symptoms," and not upon any "specific difference in the symptoms" of the two forms of the disease, suppurative and non-suppurative.

The time-honoured signs—redness of the skin, and œdema over the inflammatory mass in the right iliac fossa—are of course met with from time to time. But it may be said of them in the case of perityphlitis, as in the similar case of an empyema, that the appearance of such signs does by itself condemn the action of those who are in charge of the patient. It presupposes hesitation and delay, and the expectation of the occurrence of such gross signs of suppuration in the case of appendix-disease has cost many a life.

Fluctuation, again, is a weak reed to lean on. In the majority of cases, when the time has arrived for a surgical exploration, no fluctuation can be detected. Sometimes the pus lies too deep, being covered by adherent small intestine and even by the cæcum itself. Sometimes, though the total amount of pus may be considerable, it lies in thin layers and small pockets between the intestinal coils, so that this sign is not yielded. In many cases, however, fluctuation can be detected when the patient is under the influence of an anæsthetic, and this method of examination is most useful here, as in every form of abdominal disease.

As a rule the presence of pus can only be recognised by observing the course and daily variation of the case, and comparing it in these respects with the non-suppurative form already described.

And in many cases it is impossible upon a single examination of the case to determine whether pus is present or not, unless full details of the previous course of the symptoms can be obtained.

(a) The induration.

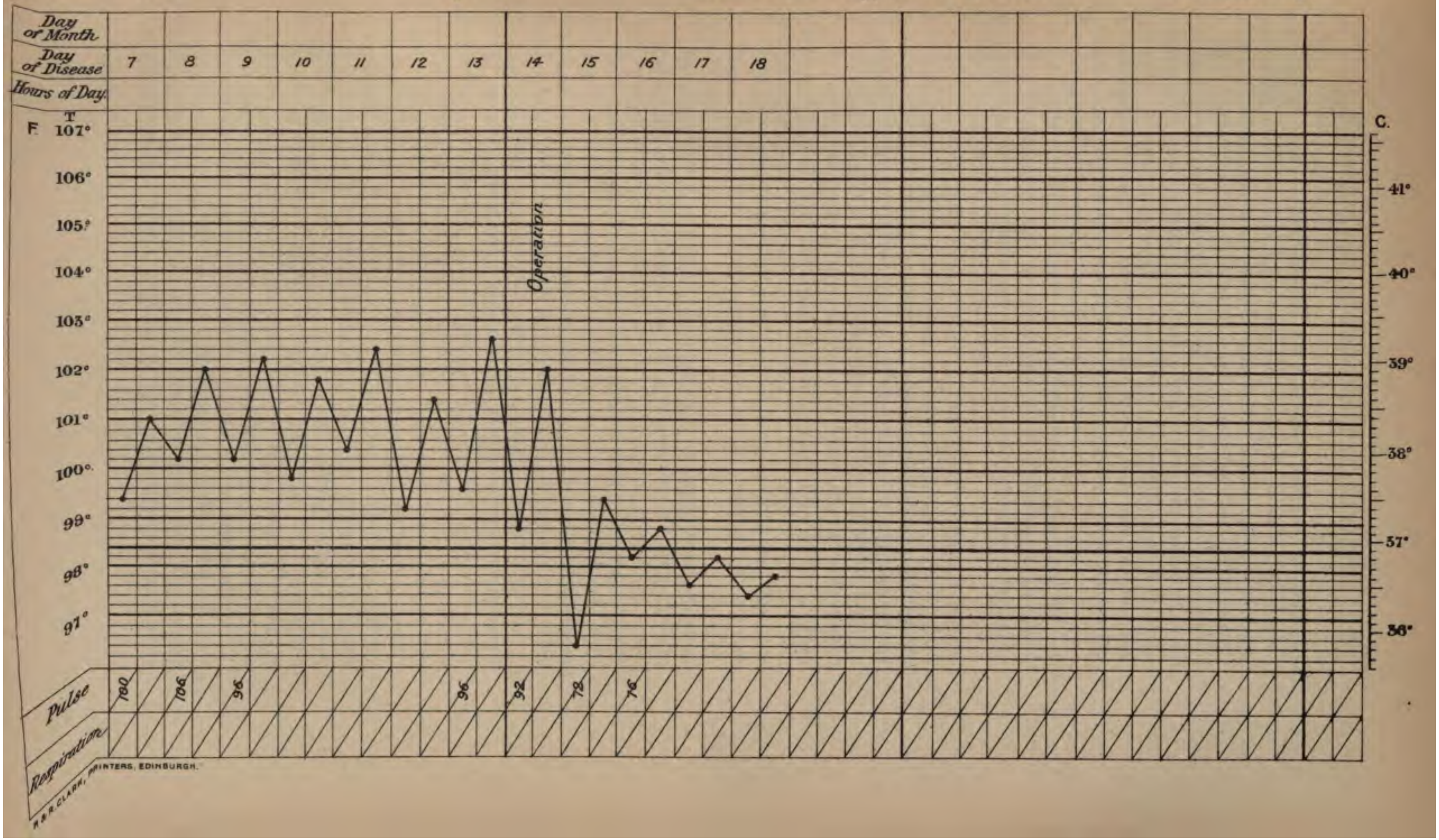
One of the most important points is the behaviour of the inflammatory mass in the groin. It has been already stated that a very large indurated mass of this kind may disappear without the formation of pus. In one case it measured four inches in all diameters, and yet complete resolution took place. Yet it is true, as a general rule, that the larger the mass the greater is the probability of suppuration. Again, the speed with which a large mass is developed is an important indication. In the non-suppurative case it is of slow formation, and several days may elapse before an appreciable size is reached. On the other hand, in some of the instances of perityphlitic abscess very large dimensions may be more quickly assumed. In the case of a boy, aged thirteen, whose symptoms appeared suddenly while drawing a truck, it was found that on the third day a dull hard mass extended up to the umbilicus from the right iliac region. This was incised on the fourth day, a large quantity of pus having a fæcal odour was evacuated, and the boy speedily recovered. The adhesions limiting the abscess in this case must have been of the slenderest description.

It appears, then, that an abscess may be fully formed as early as the third day. Fitz (*loc. cit.*) quotes a case recorded by Peckham,[77] where death occurred on the fourth or fifth day, and in the right iliac region there was found post-mortem a cavity containing nearly three pints of fœtid purulent fluid.

In such acute cases we must be guided by the formation of an unusually large inflammatory mass in an unusually short space of time.

But in most cases events are much slower. As a rule, in simple perityphlitis the mass, having attained its maximum size in six or eight days, remains stationary for a few days, losing its

Name G.R.— Age 14 Disease Perityphlitic Abscess. Index No. 8.



tenderness, and then begins to subside, and it is in this stationary period that the possibility comes before us of pus either already present or now in process of formation. Nothing in the history of the case, and no present sign or symptom, is sufficiently constant to aid in settling this question. The main indication in all such cases upon which a diagnosis of the presence of pus must rest, is either the maintenance of the inflammatory mass in a stationary condition for an unusual period of time, or the occurrence of a further increase in its size, or a further increase in its tenderness.

In other words, an important indication of the occurrence of suppuration lies in the continuance of symptoms, or in their aggravation at a time when in a non-suppurative case subsidence by resolution should set in.

(b) The fever.

In the next place, the course of the temperature must be watched, and equally important information can thereby be gained. The average duration of fever in a simple perityphlitis is roughly from six to ten days, and there is a steady decline in it from first to last. Now if the temperature in these thirty-eight cases of perityphlitic abscess is studied, in every case there is found a departure from what may be called the normal non-suppurative course.

In one case thirteen days go by, and during the last six of these days the temperature shows a tendency to rise (Chart 8).

In another case fifteen days elapse: the first seven days of this period show the usual fall to normal, the last eight days show a steady rise (Chart 9).

In most instances, then, we see some indication of the normal subsidence, but suppuration sets in, and a secondary rise of temperature begins. In other cases the temperature shows no tendency to fall, but there is continuous fever from the first day of the illness until the abscess is opened (Chart 10).

It appears, then, that the course of the temperature is an important indication. It certainly is possible, however, that pus may be present without any fever. In one of these thirty-eight patients was this the case. He was a boy, aged 14, who was seized with the usual symptoms five weeks before admission into the hospital. On admission he was greatly wasted, and lay with his legs drawn up; he presented a large hard swelling in the right iliac fossa extending round into the lumbar region, with visible veins coursing over it; and he was troubled with profuse diarrhœa. Nine days were allowed to elapse after his admission before the abscess was incised, and during this time the temperature was normal or subnormal.

Such a case as this would hardly occur at the present day, and we know nothing as to the fever during the five weeks before admission, which may have presented the characteristic features. But, with this exception, all the cases of perityphlitic abscess showed the temperature-variation indicated above.

It must be noted however, on the other hand, that fever may persist for a long period, four weeks or more, without any formation of pus. In this long course it may from time to time show a rise, which must always suggest the onset of suppuration, and still complete resolution may occur. Of this Chart 6 may be taken as an illustration.

Again, Charts 3, 4, and 5 show that a rising temperature in the course of the illness is not by itself a certain indication of suppuration.

It may be said, then, that in all cases attended by suppuration the fever does not show the usual decline, or else a secondary rise of temperature sets in. On the other hand, both these phenomena may be seen in exceptional cases of simple non-suppurative perityphlitis. So that the temperature stands to us as an important but not an infallible guide.

(c) The general condition of the patient.

In the third place stands the "general condition" of the patient as an indication of the presence or absence of pus.

It is not possible to put the meaning of this phrase into exact words. It includes the condition of the tongue as to its epithelial change and its degree of moisture or dryness; the readiness to take food; the frequency and character of the pulse; and the whole aspect of the patient, face, body, and mind.

Conclusions.

There is no infallible guide in any one of these three factors: neither in the inflammatory mass, nor in the temperature, nor in the general condition of the patient. But if all three are taken into consideration with a full knowledge of the natural course of a simple non-suppurative perityphlitis, there is little room for error in the diagnosis of abscess-formation around the appendix.

Sources of error.

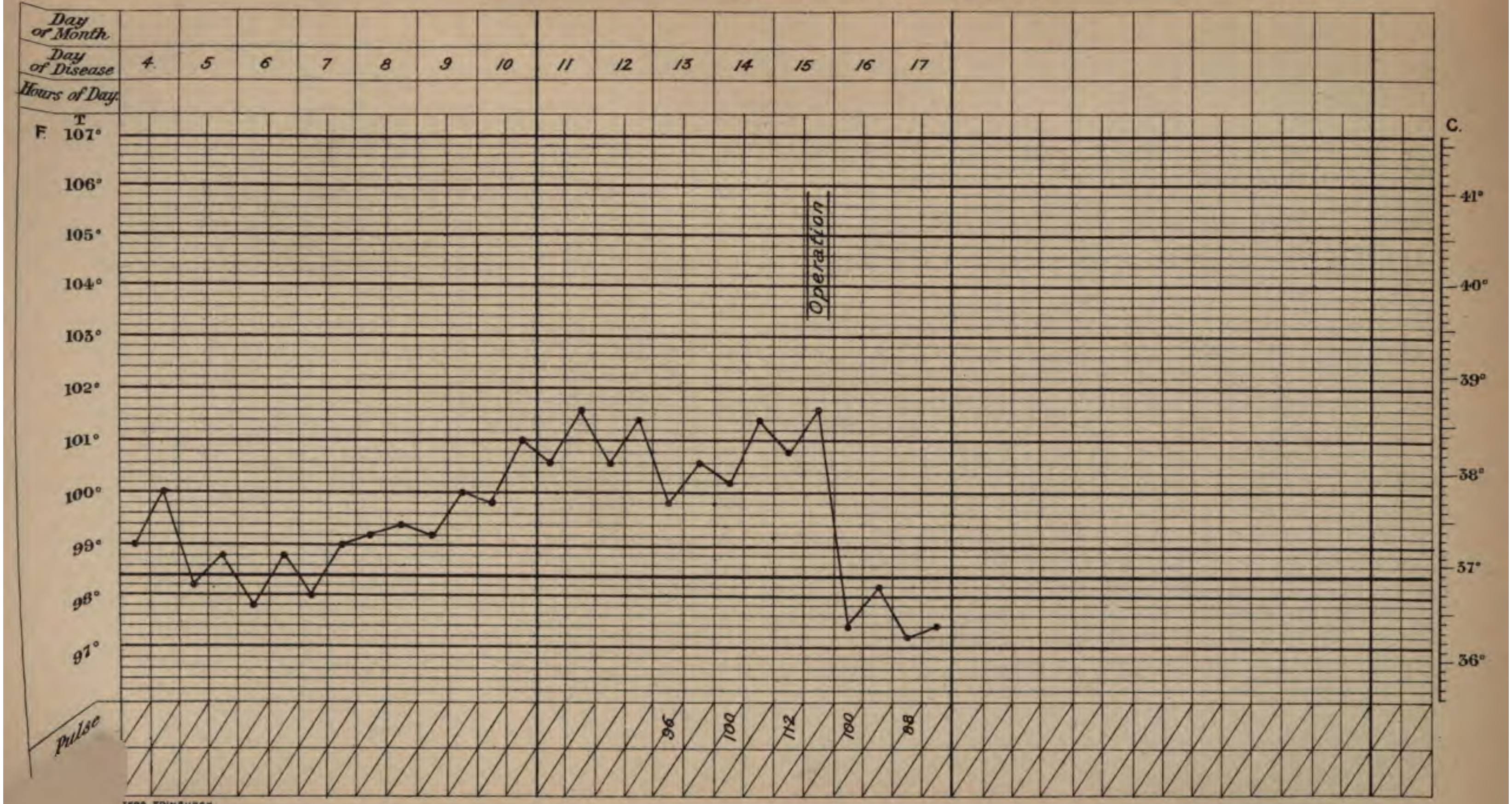
Yet it must be confessed that in some exceptional cases there is room for hesitation.

It must be borne in mind, for example, that a considerable mass may remain in the right groin for many weeks unaltered, and so occasion doubt. Exploratory operation has shown that it commonly consists in such cases not of coherent intestine but of thickened adherent omentum. An elongated roll lying above and parallel with Poupart's ligament persisted in one case for five weeks before it showed any diminution in size.

It has been already mentioned that the presence of pus is possible with no rise of temperature—an event so rare, however, in the case of appendicular peritonitis that it will hardly prove a difficulty.

More important is the knowledge that, as in the case from which Chart 6 was taken, more or less fever may persist for

Name H. W. *Age* 24 *Disease* Perityphlitic Abscess *Index No.* 9.



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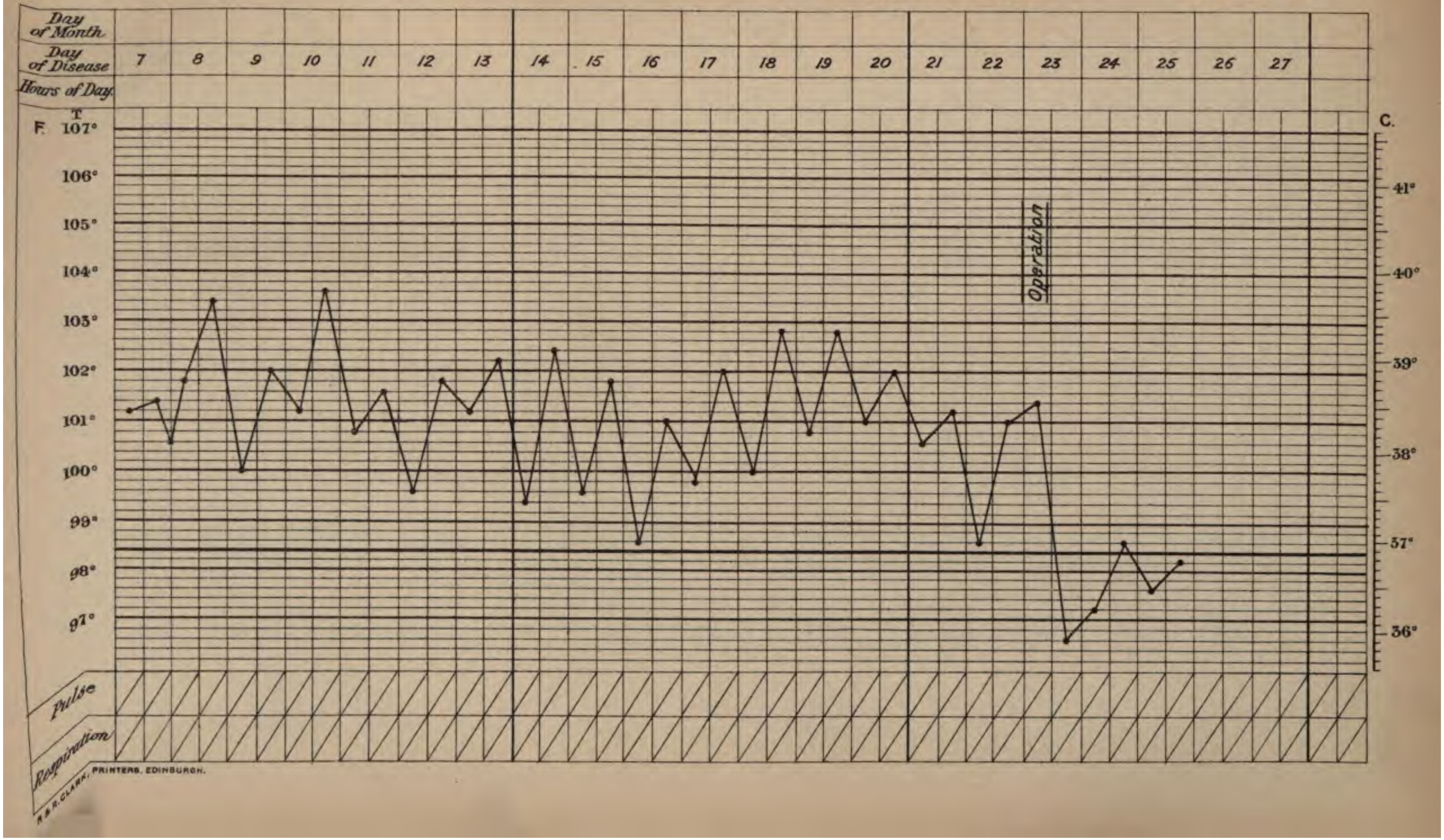
Name A. F. Age 33 Disease Perityphlitic Abscess Index No. 10.



four weeks or more, and yet recovery occur without operation. I believe that in such cases there is some small collection of pus, possibly only a few drops, deeply situated alongside of the appendix. But a continuance of fever without any tangible inflammatory mass in the groin should at once direct attention to the pelvis; for a large part or the whole of an appendicular abscess may lie within the pelvis, and it may in this way be beyond the reach of abdominal examination. An instance of this will be given later.

Again, a continuance of fever may be attributable to the supervention of one of the complications of appendix-disease, which will be considered later, such as an infective thrombosis of a branch of the portal vein and hepatic abscess, and the rare occurrence of actinomycosis of the appendix must similarly be kept in view.

It is of course important to recognise the presence of pus as early as possible, and to release it by operation without delay, but at the same time it is clear that, except in those cases (one example of which has been already given) where pus is formed at a very early date, there is no great urgency. Delay of a day or two makes no material difference, so long as the patient is at perfect rest in bed. In the early cases the limiting adhesions are soft and unorganised, so that there is considerable risk of their rupture by any sudden movement of the patient, and a consequent discharge of the abscess into the abdominal cavity. But in most instances the pus-formation is a much slower and later process, and by the time the abscess has attained a moderate size it is bounded by very strong adhesions. There is then scarcely any danger of general peritonitis except by some violent movement. Some patients walk up to the hospital and apply for admission with a large local abscess of this kind. In former days, when redness, œdema, fluctuation, and such like were awaited before operating, these cases ran a very long course unrelieved, though the abscess often discharged into the bowel.

The subjoined chart (Chart 11) shows the patience and resisting power of nature. In the case from which it was taken an abscess was allowed to lie undisturbed for upwards of seven weeks.

Abscess discharging its contents into the bowel.

Ten or fifteen years ago it was not an uncommon event that an abscess opened into the bowel. Five certain instances of this occurred in our thirty-eight cases, and all of them made a good recovery. In one case on the sixteenth day of illness as much as half a pint of pus was discharged from the rectum. The evacuation of the abscess in these cases occurred between the thirteenth and eighteenth day of the illness. An abscess may also open into the bladder.

The result.

The record in these thirty-eight cases is not a very good one, and I am conscious that it is susceptible of improvement. The prognosis in perityphlitic abscess is as a rule very favourable, but a certain proportion of deaths (perhaps 20 per cent.) cannot be avoided.

In five of the thirty-eight cases the abscess discharged into the bowel. All of these patients speedily recovered. But it is not an occurrence which is likely to recur at the present time.

Of the remaining thirty-three, ten died, and twenty-three recovered after operation, and have remained well so far as can be ascertained (except in one instance, where a recurrence of abscess occurred), although in no case was the appendix removed.

As to the ten patients who died, there was in most cases some cause for death apart from the mere suppuration. In four cases death resulted, as will be subsequently shown, entirely from obstruction of the bowels caused by adhesions. One died from a secondary abscess in the liver. One had a miscarriage and died. In two cases the local collection of pus was opened by an incision in the middle line of the abdomen, under the idea that a general peritonitis was present. It followed that the adhesions built up by nature were broken through by man before the pus could be reached and set free. This is not likely to occur at the present day.

In the ninth case the patient was seventy-six years of age. The operation was performed on the day of admission into hospital, the sixth day of the illness, and she died collapsed in a few hours.

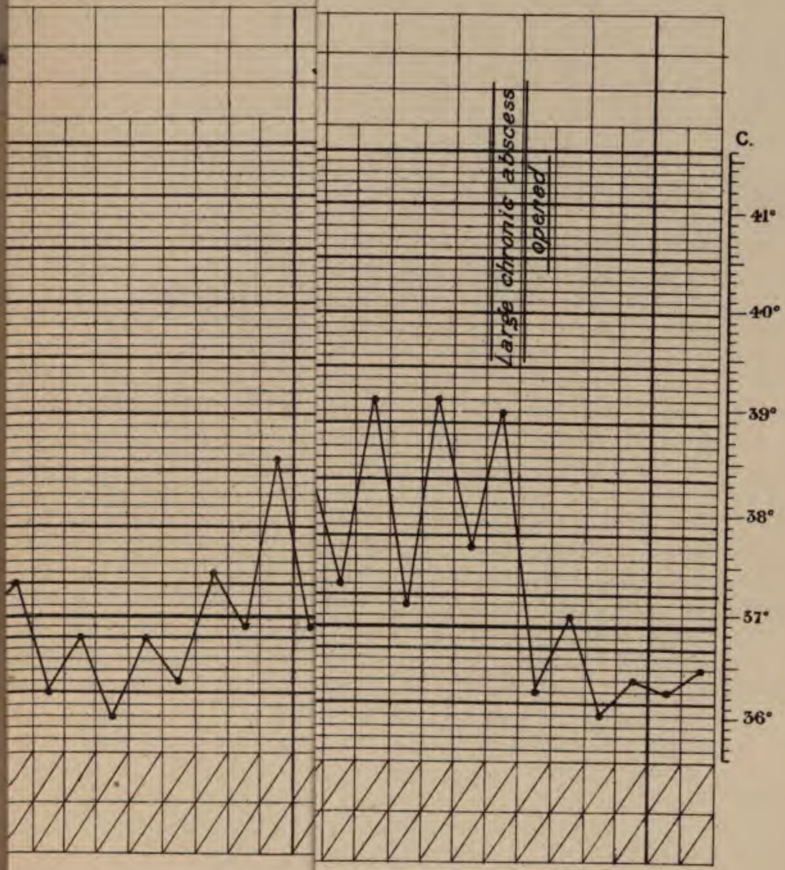
In the tenth case the patient developed the usual symptoms of local peritonitis, but stuck to his work for nineteen days. On the twenty-first day of his illness he was admitted into hospital extremely ill. The abscess was opened at once, but he died within twenty-four hours.

Position and size of the abscess.

The abscess varies much in size. In a case quoted by Fitz a perityphlitic abscess was reported to contain more than a gallon of pus, liquid fæces, and scybala. Three or four ounces are commonly met with. In some cases pus may be present in very small quantity: in one of our cases barely a teaspoonful was released, and a speedy recovery ensued. And in many cases the amount of pus is less than would have been expected from the size of the tumour.

When the collection of pus is a small one, it may be difficult to find. When several ounces are present, the small intestine which normally overlies the cæcum is pushed aside, and the abscess-cavity has the abdominal wall for a roof, and the cæcum and iliacus or psoas for a floor, with a wall of adherent bowel on the left.

When the amount is small, it may lie on the cæcum or alongside of the cæcum. The cavity is then roofed in by small intestine. In such cases, though the actual mass in the iliac fossa is a large one, the surgeon must be prepared to cut down


C.
41°
40°
39°
38°
37°
36°
Large chronic abscess
opened

upon a tumour over which there is a perfect or slightly impaired resonance.

It must be remembered, also, that the abscess-cavity is not always of a regular shape. It may exhibit processes running in different directions among the adjacent intestinal coils, though still firmly shut off by adhesions from the general peritoneal cavity. In one such case there were three processes, each of which just admitted the forefinger, and of two of these the end could not be reached.

Sometimes a process of this kind leads down into the pocket between uterus or bladder and rectum, and here a secondary pool of pus may be lying.

But the abscess may be situated entirely within the pelvis, while the iliac fossa is empty. In one remarkable case that I have seen, a patient who had had an ordinary and apparently slight attack of simple perityphlitis began after a few days of afebrile condition and apparent convalescence to exhibit a progressive rise of temperature, such as commonly warns us of the formation of pus. After a steady rise in temperature for four days, physical examination of the abdomen revealed nothing more than tenderness in the groin: there was no resistance in this region, and there was nothing abnormal to be felt on the floor of the iliac fossa. Yet at this moment there was already formed in the pelvis a considerable abscess, which was opened a few days later and found to contain exceedingly fœtid pus.

It is thus impossible in some cases to distinguish between an abscess of appendicular origin and one arising around a Fallopian tube. This is of no great moment, since an exploratory operation is necessary in both cases.

A note made by Dr. Cullingworth after vaginal examination in a case of perityphlitic abscess of this kind may be quoted: "Uterus pushed to the left by a swelling fixed and irregular, high above the vaginal roof on the right side. This mass is adherent to the uterus, and the intestine above it is distended. The swelling extends into Douglas's pouch, where a process from it can be felt adherent to the posterior uterine surface. The upper surface of the mass cannot be exactly mapped out, owing to the abdominal distension. Nothing abnormal on the left side. Diagnosis of inflammatory mischief around either the right uterine appendages or the appendix."

Sometimes however the pelvic abscess may be only part of a larger collection, which may extend across the lower part of the abdomen from ilium to ilium. Such a collection of pus may be firmly roofed in by matted intestine and omentum, and may yield complete dulness on percussion from umbilicus to the symphysis and from the right to the left iliac spines.

It is rare to find the appendix obtruding itself upon our notice after evacuating a perityphlitic abscess, and in fact it is very difficult to find it without making a search which will endanger the integrity of the limiting adhesions.

In some cases, which will be understood from the variability in the position of the appendix, the collection of pus may lie on the outer side of the cæcum. Bryant (*loc. cit.*) mentions three cases in which the tumour had been located from the first between the lower ribs and the anterior superior spine of the right ilium. In these cases the incision was made above the crest of the ilium, the pus lying outside the meso-colon.

Extra-peritoneal suppuration.

A belief is still met with in some quarters that the suppuration in some of these cases is outside the peritoneal cavity. The origin of this belief can be understood from the writings of the beginning of this century, and it has been fostered by the erroneous account of the arrangement of the cæcal peritoneum, which has been received without question until recent times.

It has been already shown that the appendix is always an intra-peritoneal organ, as also is the cæcum.

It is, however, possible for an extra-peritoneal suppuration to arise from disease of the appendix. It sometimes happens, either in neglected or in very virulent cases, that the peritoneum, which covers the iliacus muscle and forms the floor of a perityphlitic abscess, may become secondarily necrosed and perforated, so that pus may obtain entrance into the retro-peritoneal tissue and burrow therein. The same process occurs occasionally in pelvic abscess due to disease of the Fallopian tubes.

As Fitz puts it, "With perforation of the parietal peritoneum may occur extensive necrosis, purulent and fæcal infiltration of the abdominal walls. Within three weeks the iliac muscle may be destroyed and the ilium be bared. The course of the psoas and iliacus may be followed into the thigh, and extensive and deep-seated destruction of tissue with fæcal infiltration be present in this region. The pus may extend through the obturator foramen, forming a deep-seated abscess of the hip and thigh, and may enter the hip-joint."

Further, when the appendix is adherent to the iliacus-peritoneum, a perforation of it on the adherent side may lead directly to inoculation of the retro-peritoneal tissue, so that a large abscess may form in the iliac fossa with little or no affection of the peritoneum.

Two cases recorded by Kelynack (*loc. cit.*) seem to be fairly explained in this way. The majority of supposed examples of this kind are, however, founded on error, and the parts as found post-mortem are sometimes so surprisingly altered and confused with sheets of tough membrane interposed between pockets of pus that such an error is no matter for wonder.

Lumbar abscess.

It has been shown that the appendix lies more or less behind the cæcum in some 26 per cent. of all bodies, and that in 6 per cent. it runs vertically upwards in this position, occasionally reaching the right kidney.

It is quite possible, then, in some of the cases of unexplained abscess in the lumbar region on the right side that the appendix may be the cause of the suppuration. This origin has often been suggested. Some two or three cases of abscess of this kind are admitted every year into St. Thomas's Hospital. They are attributed to bone-disease, or are called "perinephric." They commonly recover rapidly after evacuation of the pus. The following instance is certainly suggestive of an appendicular origin.

CASE III

Lumbar abscess containing fæcal matter: operation: recovery.

E. S., a woman aged 30, was seized one afternoon with sudden pain in the epigastrium, spreading subsequently all over the abdomen, accompanied by frequent vomiting. She had enjoyed good health up to this time. On the next day the pain abated somewhat, but became localised in the right flank, mounting later to the costal margin. The vomiting continued.

She was admitted into hospital on the fourteenth day of the illness. She was then suffering considerable pain, which was most intense at a point midway between the crest of the ilium and the costal margin on the right lateral aspect of the abdomen. There was a certain induration all over the right flank, and over the situation of the kidney the skin was reddened and slightly œdematous. The rest of the abdomen was natural. The temperature varied between 101 and 104·2.

An incision was at once made over the kidney behind. About half a pint of fæcal-smelling pus was evacuated, and in the pus I discovered two small soft pellets of fæcal matter. Recovery was quick and perfect.

Abscess elsewhere in the abdomen.

Three interesting cases of perityphlitic abscess on the left of the middle line of the abdomen are recorded by Fowler (*loc. cit.*) They all proved fatal, so that their nature is beyond doubt.

In the first case a woman aged 35 was seized with sudden pain all over the abdomen, which subsequently settled in the epigastrium. In forty-eight hours she was collapsed, the only physical sign being some tenderness on the outer border of the left rectus at the level of the umbilicus, and she died seven hours later. It was found post-mortem that the cæcum lay on the promontory of the sacrum, with the appendix curled upwards and outwards under the left rectus: there was a large perforation in it, without any concretion, and there was a general septic peritonitis.

The second case occurred in a lad aged 19. There was much the same history as in the first case, but a definite tumour appeared on the fifth day of illness under the left rectus. This was incised, and sero-purulent fluid released. It was subsequently found that the cæcum lay under the umbilicus and the appendix was gangrenous: no concretion was discovered.

The third case occurred in a boy aged 16. In him there developed with severe symptoms an area of tenderness, 3½ inches in circumference, under the left rectus, commencing at the umbilicus and extending downwards. It was found that the cæcum and a gangrenous appendix lay under the umbilicus.

No certain case of this kind has occurred in our series. But I have recently had a woman aged 38 under my care who may have been suffering from this condition. She was admitted into hospital with a visible swelling, 2½ inches in circumference, partly above and partly to the right of the umbilicus: the skin here was reddened and œdematous. She had been taken ill suddenly some three weeks before with general abdominal pain, vomiting, and fever. The abscess was at once opened, the pus had a strong fæcal odour, and actual fæcal matter continued to be discharged in small amount for several days. No attempt was made to explore the abscess further, and it slowly closed up, leaving the woman perfectly well.

COMPLICATIONS AND SEQUELÆ

Intestinal obstruction.

Of all the complications and after-effects of perityphlitis and perityphlitic abscess, obstruction of the bowels stands in the front rank. In some cases it is the actual cause of death, and in many cases it persists in slight degree through a long period of convalescence. Considering its gravity, it is a complication which has received scanty attention.

It has been already mentioned that in four of our ten fatal cases of perityphlitic abscess the real cause of death was intestinal obstruction. How far this is true may be judged from the following brief notes.

CASE IV

Perityphlitic abscess: adhesion of ileum to abdominal wall: obstruction: distension, ulceration, and perforation of ileum, resulting in general peritonitis and death.

J. T., a lad aged 18, had an attack of pain in the right groin five years ago, following a blow from a fist. His present illness began six weeks before admission, with pain and tenderness in the same region, constipation, and vomiting. The week before admission he suddenly became much worse. He was admitted suffering from severe general peritonitis, and died three days later

The interpretation of this history is clear from the post-mortem condition. There was found an abscess in the right iliac fossa, evidently of long standing (six weeks), and limited by strong adhesions: in this abscess lay the appendix. Among these adhesions, firmly fixed to the abdominal wall, was a coil of small intestine. This was empty and collapsed, while the portion above was much dilated, and showed numerous distension-ulcers, at one of which perforation had occurred.

So that in this case a patient, being left for six weeks with a perityphlitic abscess, dies from general peritonitis, which is due not to disease of the appendix but to a secondary distension and perforation of the ileum.

CASE V

Recurrent perityphlitis: two small pockets of pus around the appendix: right iliac fossa filled with firmly matted intestine: intestinal obstruction, distension, and ulceration of ileum.

T. H., a man aged 27, was admitted into hospital for a seemingly mild attack of perityphlitis, which was his third attack in twelve months. He was admitted on the fifth day of his illness. He had slight fever, constipation, and vomiting. His abdomen was slightly distended, and in the right iliac region there was tenderness and a sense of rounded resistance. Although the physical signs were not grave, he seemed unusually ill.

In a few days it became clear that he was suffering from intestinal obstruction. That the obstruction was due to matting of intestine in the iliac fossa around the appendix was fully realised, but an operation was thought to be useless. As the distension of the abdomen increased, his sufferings were very severe, and the relief obtained by persistent puncturing of the intestines was, as usual, so temporary as to be useless. Death, which occurred on the twenty-sixth day of the illness, was not unwelcome.

It was found at the post-mortem examination that the right iliac fossa was occupied by a mass of coherent intestine, and the adhesions were too strong for tearing, and too intricate for dissection. Among them lay two loculi of light-green glairy pus, about two drachms in each. The whole of the small bowel was much distended, and the lower part of the ileum exhibited six distension-ulcers.

CASE VI

Perforation of appendix: perityphlitic abscess with adhesions: intestinal obstruction and death.

E. L., a lad aged 18, had a slight attack of perityphlitis five months before his present illness. He was admitted on the eighth day of illness with obvious intestinal obstruction. He was emaciated and worn, with sunken eyes: his belly was distended, and showed peristaltic movements of visible coils of bowel: constipation was absolute. He died three days after admission.

In this case the fatal adhesions were probably formed in the attack of perityphlitis five months before admission, and possibly also a small abscess, found around the tip of the perforated appendix, arose at the same time.

The post-mortem examination showed that the obstruction lay in two feet of ileum which were collapsed and firmly glued to the right side of the pelvis.

The fourth case was under my own care. The obstruction and its particular cause were clear enough, and the abdomen was explored on the day of admission, with the idea of relieving the obstruction, but the patient went the way of the others.

CASE VII

Necrosis and perforation of the appendix: perityphlitic abscess: adhesions: obstruction and death.

E. T., a boy aged 16, was seized with pain in the right iliac region while lifting some heavy luggage. The usual symptoms of perityphlitis set in, and then he began to improve. But he was seized with vomiting and paroxysmal pain in the third week of his illness.

After ten days of complete obstruction he was admitted into hospital in the usual deplorable condition, with distended belly, visible peristalsis, fæcal-smelling vomit, and exhaustion.

Nothing could be done at the operation beyond release a few pockets of pus and make an artificial anus into the small intestine. Some of the softer adhesions were torn through, but one portion of the ileum was so firmly adherent to the wall of the pelvis that it could not be set free.

It was found at the post-mortem that there was a collection of two ounces of pus inside the pelvis on the right side, and in this lay a laminated fæcal concretion. The appendix ran down the pelvic wall, and its tip had disappeared, leaving a gaping black orifice. The ileum was flattened and firmly adherent inside the pelvis, climbing about on its walls like ivy on a stone. The rest of the small bowel was much distended.

It appears from these cases that intestinal obstruction may close the scene in cases of perityphlitic abscess which are not otherwise severe.

Mild degrees of obstruction are not uncommon in perityphlitis without suppuration. It has already been pointed out that in some such cases the pain may even at an early period assume a paroxysmal character, and may be attended by periodical distension of the adjacent portion of the ileum.

In some cases, as adhesions form, this paroxysmal pain and violent peristalsis may continue for many weeks. In the majority of them these symptoms slowly pass away. In the case of a man aged 30, who had had two previous attacks of perityphlitis, a third slight attack left a fairly hard mass of the size of a hen's egg in the right iliac fossa. For the next four months he had innumerable attacks of spasmodic pain in this position, with borborygmus, abdominal distension, and occasional vomiting. He ultimately recovered, and the iliac mass melted away.

Another example is the case of a boy, aged 15, who had had an attack of general peritonitis, succeeded after a lapse of eighteen months by two attacks of local peritonitis in the right iliac region. On the subsidence of these latter attacks he remained for six months liable to attacks of intermittent and incomplete obstruction, in one of which I saw him. He ultimately recovered, contrary to my expectation.

Pylephlebitis and hepatic abscess.

Hepatic abscess is another complication which may ensue in any case where a collection of pus remains for any length of time in the peritoneal cavity. And there can be no doubt that it is in all instances due to an infection of the veins in the wall of the intestine at the diseased spot, with subsequent extension of the infection to the liver.

Only one instance occurred among the thirty-eight cases of perityphlitic abscess here recorded.

CASE VIII

Perityphlitic abscess followed by abscess of liver: death.

A. F., a man aged 33, was seized with the usual symptoms of perityphlitis. He was admitted into hospital on the seventh day of his illness, and he then presented a mass the size of a man's fist in the right groin. In a few days this mass increased somewhat in size, and from this and from the continuance of the fever it became clear that suppuration had occurred. The usual incision in the right semilunar line was made, 4 oz. of fœtid pus evacuated, and the cavity washed out and drained. For three or four days the usual marked benefit resulted, and he remained free from fever.

Then the temperature began to rise occasionally in the evening, frequent sweatings occurred, and the patient seemed to be growing thinner. At first one could only conclude that the abscess-cavity was not thoroughly drained. A counter-opening was made in the flank, and the parts searched for any remaining pocket of pus.

But the fever grew apace and assumed a definite hectic character, rigors occurred from time to time, and emaciation was rapid.

Finally, five weeks after the original evacuation of the abscess, the explanation of the unusual course of the case appeared in a small rounded swelling that began to show itself from under the costal margin immediately to the right of the ensiform cartilage. This swelling grew rapidly in size. It was incised a day or two later, and half a pint of pus was evacuated. The patient lingered on for two months longer. A post-mortem examination was refused.

It is not a common condition, but many cases have been recorded. As a rule, the suppurative hepatitis is the result of an extension of an infective thrombosis from some radicle of the portal vein up through the main trunk of that vein into its divisions in the substance of the liver. Fitz found this to occur in eleven of 257 cases of perforative appendicitis. The infection may travel from the portal trunk down the splenic vein. Sir Dyce Duckworth[66] mentions a case where abscess both in liver and spleen resulted from perforation of the appendix.

It is not only in cases of suppuration around the appendix that this typical pylephlebitis with ramifying channels of suppuration in the liver occurs. In a case of this kind recorded by Payne[67] the infective thrombosis of superior mesenteric and portal veins resulted from ulceration of the mucous surface of the appendix without any recent peritoneal inflammation around it.

A similar case is mentioned by Janeway.[68] There was "an old ulceration of the appendix, which was glued to the brim of the pelvis: there was a spot of local inflammation, no larger than the end of my finger, pressing on the ileo-colic vein: this and the portal vein were thrombosed, and there were multiple abscesses in the liver."

The condition of the liver in these cases of complete phlebitis is such that no hope of relief by surgical interference can be entertained. It is permeated throughout by channels of pus, with multitudes of small suppurating foci in every part of it.

In some cases, however, a single hepatic abscess is formed as the result not of a true portal phlebitis but of an infective embolism. Infected clot is formed in a minute vein (for example, in the appendix-wall), and this is subsequently detached and lodged in the liver. This embolic abscess is as amenable to surgical treatment as a dysenteric abscess. In seems probable that the hepatic abscess in Case VIII., from its size and from the absence of any evidence of general enlargement of the liver, should be regarded as one of this kind.

Payne (*loc. cit.*) records a case which seems to have been of this embolic nature. There was found in the appendix a pin, around which a concretion had been formed, but there was no sign of acute inflammation of its serous or mucous coat. There was no peritonitis. The large portal branches and the main vein were healthy. Yet the liver contained a large abscess, with which two or three smaller pockets communicated.

The condition of the vein in the appendix-wall likely to lead to hepatic abscess in this way has been illustrated in Figures 14 and 19.

It is to be noted also that the pus in the hepatic abscess of Case VIII. was as fœtid as was the pus in the perityphlitic abscess. It has already been mentioned that the bac. coli communis has been found in the pus in such a case both in the perityphlitic and in the hepatic abscess (Hodenpyl).

In three cases recorded by Gendron[69] hepatic abscess arising from disease of the appendix burst upwards through the diaphragm.

Pyæmia.

In some rare cases the infective process passes beyond the liver, and an infection of the systemic circulation ensues. Duckworth (*loc. cit.*) quotes three cases. In one there was an abscess of the brain as well as one in the liver. In another there were found purulent infarcts in the liver, and several abscesses in the lungs and brain. In the third case there were abscesses in pancreas, liver, lungs, and brain.

Fæcal fistula.

In five of our thirty-eight cases, as has been mentioned, the perityphlitic abscess discharged into the bowel. All of these recovered nearly as quickly as if the abscess had been opened by a surgeon.

In three other cases, after the abscess had been evacuated by the usual incision, definite fæcal matter was found in the dressings for several days. All these recovered quickly.

It is not wise in any such case to interfere: the fistulous opening will be sealed and closed as the abscess-cavity heals.

In the following case the abscess burst externally before admission into the hospital, leaving a fæcal fistula.

CASE IX

Chronic perityphlitic abscess, due to perforation of appendix by a concretion: persistent umbilical fistula.

G. W., a boy aged 13, gave a history showing that three months before admission he had an attack of perityphlitis from which he did not recover completely: six weeks later a lump, which had persisted throughout, began to discharge at the umbilicus. On admission he presented a fairly well-defined triangular mass in the abdomen, the base lying upon the inner half of the right Poupart's ligament and the apex being at the umbilicus, where there was a button of granulation-tissue emitting a scanty fæcal discharge. This sinus was opened up and found to lead into a circumscribed abscess-cavity, in which lay a fæcal concretion of the size and shape of a bean. He recovered completely, though the induration persisted for some months.

Occasionally a secondary perforation of the cæcum occurs at one or more spots. This was probably a common event when it was the custom to wait for fluctuation as a sign of abscess, but it may be met with in cases of not very long standing. In a case recently under my care, in which a large perityphlitic abscess filled the pelvis and right iliac fossa, the cæcum presented three separate perforations. Mr. Ballance, who operated, decided that nothing could be done but bring the cæcum up to the surface and fix it so that the fæcal matter could only escape externally. The edges of the perforations were too soft and rotten to allow of suturing. Death took place two days later, but no general infection of the peritoneum had occurred.

Treves[70] recommends that no immediate attempt be made to close a cæcal perforation by operation, owing to the condition of the bowel-wall.

Inflammation of psoas and iliacus.

As might be expected after the long continuance of an abscess lying upon the iliacus or psoas, or both, there may appear on recovery considerable stiffness in using the right leg. This is seldom of any great importance. In one case of simple perityphlitis it was well marked. The patient had had rather a long illness, lying for some time with his legs drawn up, and when he was in other respects perfectly well it was found that the right thigh could not be completely extended on the pelvis. This condition persisted when under the deep influence of chloroform.

Such a condition is probably dependent on a definite interstitial inflammation of the muscle. The specimen shown in Fig. 20, in which such an inflammation is plainly seen, was taken from the iliacus of a patient who died from a perityphlitic abscess.

Œdema of the right leg.

A persistent œdema of the right leg is occasionally met with. Osler (*loc. cit.*) mentions a case. It should probably be explained by the contraction of inflamed retro-peritoneal tissue around the external iliac vein.

Hæmorrhage.

It has happened that a blood-vessel has been opened up by an invasion of its walls. Fowler (*loc. cit.*) narrates an instance of this kind, where a man aged 57 had an attack of local perityphlitis consequent upon a gangrenous condition of the appendix. Laparotomy was performed, and the wound-cavity was thoroughly cleansed and drained. Two days later, while the wound was being dressed, a gush of blood followed the withdrawal of the gauze drains. The hæmorrhage was temporarily controlled, but he never rallied from the loss of blood.

Bryant[71] records a case of fatal hæmorrhage from ulceration of the deep circumflex artery.

Powell (quoted by Fitz) met with a case where the appendix was adherent to the internal iliac artery and their cavities communicated. The colon and cæcum were distended with gas and dark blood.

Pleurisy.

In two cases (out of 190) a definite right-sided pleurisy developed in the course of a simple perityphlitis. This complication is more frequent and more severe, as will be seen later, in cases of general peritonitis. It is of interest, now that the bac. coli communis has been often found in the lung-complications accompanying intestinal diseases.

Appendicitis in

The occurrence of inflammation of the appendix when that organ happens to lie in the sac of a hernia has been often *hernial sac.*

described. Unless this condition is known to occur, the symptoms are likely to be gravely misleading.

In 1848 Thurnam [72] recorded such a case. A male, aged 56, had long had an incomplete hernia in the right inguinal canal, and wore a truss. The tumour in the canal began to increase in size, until it was as large as a hen's egg, and it became very painful, though there was little or no constitutional disturbance. It was leeched, and calomel was given. In a fortnight's time it had occupied the whole right side of the scrotum. Ten days later fluctuation was detected: the abscess was opened, and half an ounce of fæcal pus with some gas was released. Seven days later a second point of fluctuation was detected: on incising this, some more pus was discharged, and with this there came

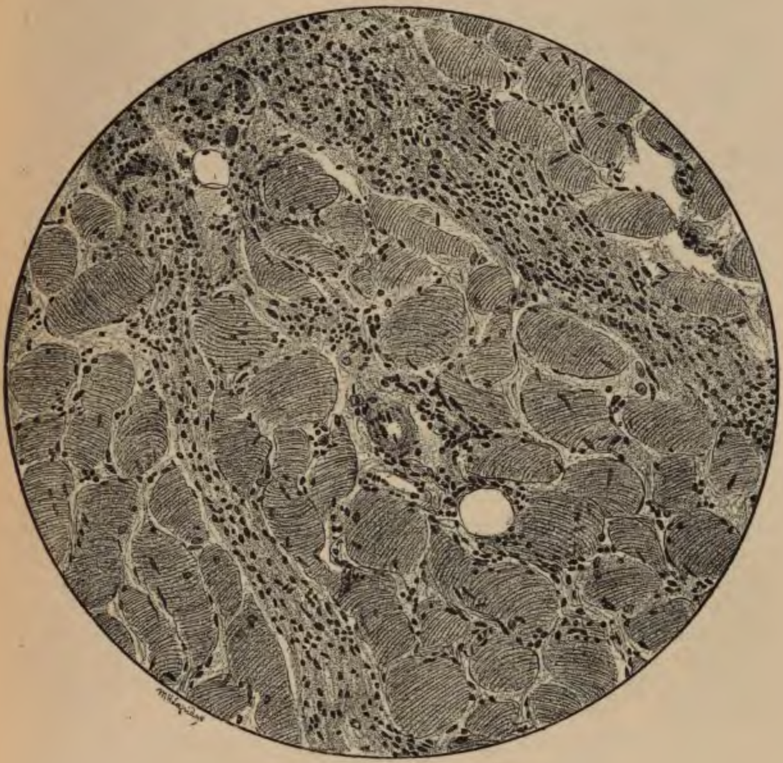


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FIG. 20.

Section of iliacus muscle in a case of perityphlitic abscess.

away a triangular piece of bone with sharp edges (part of a mutton chop). He recovered completely, after a long illness.

A month afterwards he died by an accident. The vermiform appendix was found to have twice the usual length, and three times the usual size, and it was much thickened. The lower three fourths of it lay in the inguinal canal, and its tip was firmly adherent to the bottom of the canal by an elongated firm white band.

Rand[73] mentions a case where an old irreducible femoral hernia became the seat of a phlegmonous inflammation: incision into it showed the presence of the inflamed appendix with an encysted sero-purulent collection.

Perityphlitis and pregnancy.

It is a serious matter when a pregnant woman develops even the local form of peritonitis around the appendix. It seems that in most cases the uterus discharges its contents.

In one of our fatal cases of perityphlitic abscess this at any rate in part contributed to death. In one of our cases of general peritonitis (to be subsequently described) a miscarriage occurred.

Fowler (*loc. cit.*) met with four instances of this kind in 143 cases. "All four developed septic peritonitis before coming under observation. In two abortion took place prior to operation, while in the others the uterus emptied itself after the operation. All four died, the abortion not necessarily hastening but rather heralding the fatal issue."

Appendicular colic.

There have been described from time to time cases where a violent pain has occurred in the right iliac region, the kind of pain that is commonly spoken of as "colic," and yet on operation no sign of peritonitis has been found. The term "appendicular colic" was originally used in a vague way to indicate severe pain in the region of the appendix. But it has lately been definitely adopted to indicate a spasmodic pain produced by a violent peristaltic contraction of the appendix itself, in the effort to expel a concretion or to eject mucus past an incomplete stricture.

Binnie[74] has written of this. Lately Jessop[75] has described two cases, in both of which he excised the appendix in periods of quiescence, and found no signs of past peritonitis.

More evidence is required before the theory of a true appendicular colic can be established. Considering the great frequency of fæcal pellets in the appendix and of strictures of its lumen, such a colic should be frequently met with, but it is certainly a rare occurrence. The mere absence of adhesions about the appendix is no proof that peritonitis has never occurred in this region, and I am sceptical as to the occurrence of any severe pain about the appendix which is not accompanied by fever.

SOME CONDITIONS RESEMBLING PERITYPHLITIS

It is hardly necessary to enter at length into the subject of conditions which may be confounded with perityphlitis, or into the differential diagnosis. But in a certain number of cases, where apparently a simple perityphlitis or a perityphlitic abscess comes on with the usual mode of onset and the usual train of symptoms, the subsequent course of the case makes it clear that we have not to deal with simple disease of the appendix. It is possible that the presence or absence of McBurney's tender point may prove of service here.

Actinomycosis.

The case of actinomycosis recorded by Ransom has been already mentioned. Here it was only after the unusually long continuance of symptoms that suspicion was aroused as to the nature of the disease.

Pelvic peritonitis.

Again, it may be very difficult to distinguish between peritonitis arising from disease of the appendix and the pelvic peritonitis of women. This difficulty has been already mentioned as dependent on the variability in the position of that organ.

Malignant disease of the cæcum.

Finally, carcinoma of the cæcum may form a hard mass in the right iliac region, which in itself may closely resemble the perityphlitic induration. The history of the case as a rule leaves little room for doubt in the diagnosis.

In a few cases, however, attacks of peritoneal inflammation around the new growth may occur, and the resemblance to appendicular disease is then very close.

As an example may be taken the case of a patient, aged 38, who was admitted into hospital suffering apparently from a simple perityphlitis. The right iliac mass subsided to a certain extent, but a hard nodulated tumour remained. Though the acute symptoms entirely disappeared, yet it was suspected that this remnant of the tumour was a new growth on account of its extreme hardness (which was much greater than that of the omental masses sometimes left after attacks), and on account of the progressive emaciation. He was discharged from hospital at the end of a month, but was kept under observation. Four months later he was again admitted. The tumour had grown rapidly, and he died a month afterwards. There was found to be a carcinomatous mass almost replacing the cæcum.

In some cases what appears to be a definite perityphlitic abscess develops around a carcinomatous growth of the cæcum. A knowledge of the occurrence of such a condition should lead to care in giving a prognosis, where the patient is past middle life and shows emaciation or an unusual degree of illness.

CASE X

Carcinoma of cæcum: perforation: formation of a local abscess: operation: death.

A. N., a woman aged 54, was suddenly seized with pain in the right groin seven months before admission. The pain continued throughout the illness, but she had never taken to her bed. There was no vomiting at any time, and the bowels acted fairly regularly. About a fortnight before admission the pain increased in severity.

On admission she was thin and anæmic. The tongue was dry, red, and raw; the temperature 101·4, the pulse 108. The greater part of the abdomen was natural, but there was a firm tender mass the size of a hen's egg lying above the outer part of the right Poupart's ligament, and extending slightly into the flank. Four days later, the tumour having increased somewhat in size, a severe rigor occurred, the temperature reaching 105·2 (Chart 12). For the next three days the temperature showed a wide daily variation, the induration and tenderness extended farther into the flank, and the patient became extremely ill. There was no reddening of the skin, and no œdema or fluctuation.

An incision was then made obliquely above and internal to the anterior superior spine of the ilium, and several ounces of fæcal-smelling pus evacuated, in which were found two grape-stones. No sign of malignant disease was discovered by the surgeon in the abscess-cavity, although its existence was suspected from the age of the patient and from the long irregular course of the illness.

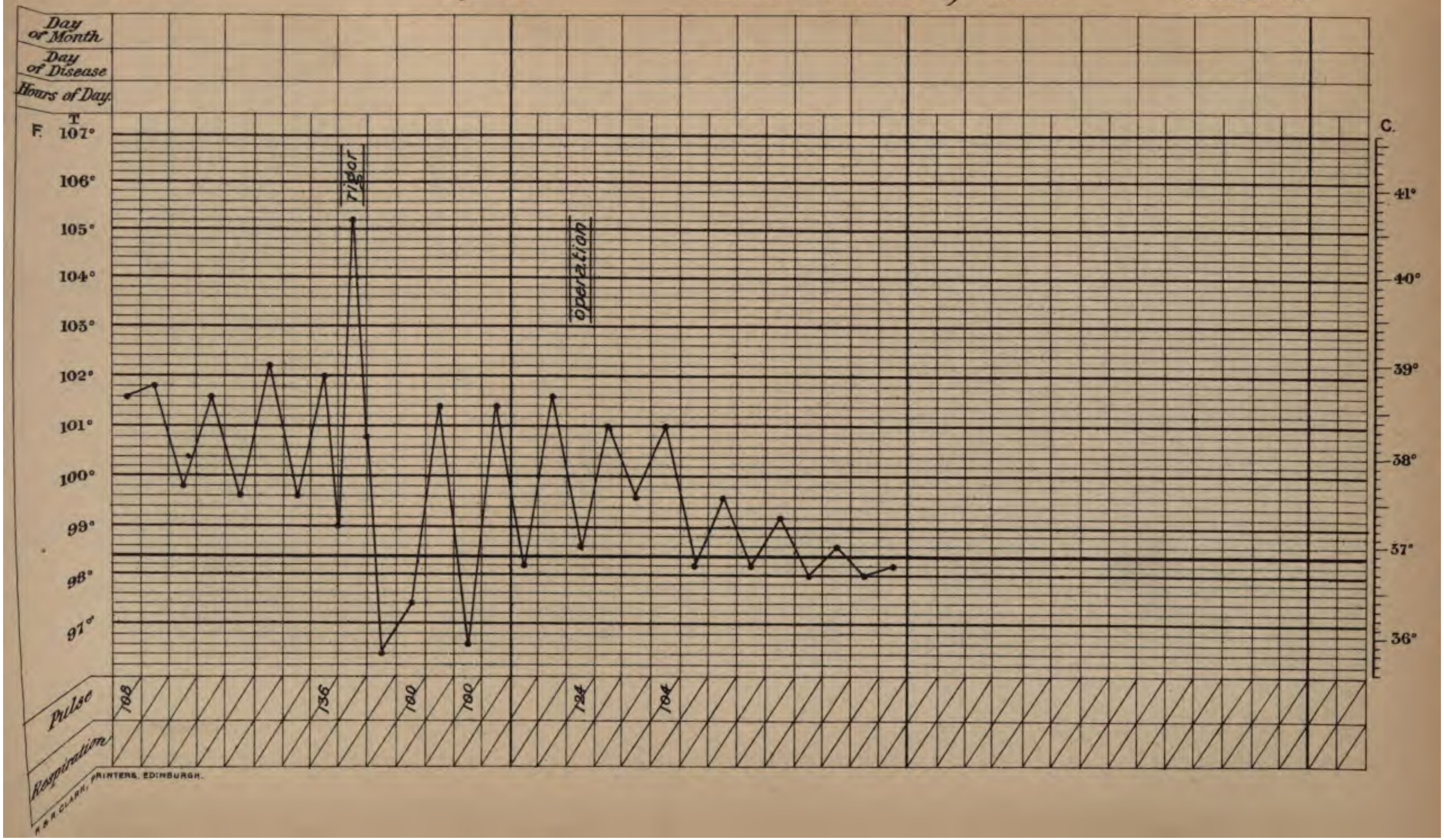
The temperature became normal, and remained so. The discharge diminished rapidly, though the perforation was known to be still open, as eight days later another grape-stone made its appearance. But she sank rapidly, and died three weeks after the operation. At the time of death there remained apparently nothing in the right iliac fossa but a little thickening, and yet there was found at the post-mortem to be a flat plate of growth in the cæcum near the valve, and it was in the floor of the carcinomatous ulcer that the perforation had occurred. The appendix was healthy.

(C) GENERAL PERITONITIS

The conditions upon which a general peritonitis ensues have been shown to be chiefly (1) the presence of a concretion and its destructive effect upon the wall of the appendix, and (2) an infective inflammation of the tissues of the wall commonly associated with necrosis.

General peritonitis arising from disease of the appendix is an exceedingly fatal form, but it is difficult to determine the exact rate of mortality. In the first place, a certain number of cases show for some days evidence of general peritoneal inflammation, and as this subsides a localised inflammatory mass can be felt in the right iliac fossa. Now there may be different opinions as to what group of symptoms warrant the diagnosis of a general peritonitis. In the description here given, the signs

Name *A. N.* Age *54.* Disease *Carcinoma of Cœcum* Index Nº 12.



R. & R. Clark, Printers, Edinburgh.

on which a diagnosis of general peritonitis is based are marked abdominal distension, general abdominal pain and tenderness, and complete loss of abdominal respiratory movement.

If this criterion of a general peritonitis be accepted, thirty-six cases occurred in our series of 264. Of these thirty-six cases, nine recovered, and all of these, as the general abdominal distension subsided, revealed a localised mass in the right iliac region, which thus certified as to the appendicular origin. The remaining twenty-seven died, whether the abdomen was opened and washed out or not.

It must be said, also, that in the period during which these thirty-six cases were under treatment in the hospital, three cases of acute general peritonitis in young subjects were admitted. These recovered, and when they recovered no sign of disease in the right iliac region could be detected. Consequently, though it is not unlikely that they were of appendicular origin, they are not here included.

Though these thirty-six cases can thus be divided into two classes—viz. nine who recovered and twenty-seven who died—it is not right to draw any distinction between them from a clinical point of view. On the whole, of course, the symptoms were less severe in those who recovered, and more severe in those who died, but among the recoveries are instances where the symptoms were quite as grave as in any of the fatal cases. And it may be certainly said that, whether they recovered or whether they died, they all presented some degree of that general poisoning which is probably the one and only cause of death in peritonitis.

There is, however, one distinction between the two classes, which must be emphasised as being of some use in estimating the chances of life in any individual suffering from general appendicular peritonitis. This distinction rests on the occurrence of previous attacks. Of the twenty-seven patients who died, three only had had any previous peritoneal inflammation around the appendix, while of the nine who recovered four had previously suffered from one or more attacks of a local peritonitis.

It may be said that this difference in favour of the patient who has had previous inflammation in the right iliac region is due to the formation in these previous attacks of adhesions serving to limit a subsequent trouble. But I think it is far more probable that the explanation lies in a change in the peritoneum itself apart from adhesions. The previous attacks of inflammation will have rendered it more fibrous, denser, and less absorbent.

Whatever the explanation may be, in any case of general peritonitis supposed to arise from disease of the appendix a history of previous attacks must be put on the right side of the balance in making a prognosis.

The onset. In all but four of these thirty-six cases the onset of the peritonitis was absolutely sudden and severe. There is no warning, and seldom any exciting cause. In one case a boy was jumping and romping when the initial pain seized him, and his appendix was almost rent in twain, while a concretion passed through into the abdominal cavity. In another case a large meal of tinned salmon immediately preceded the onset of symptoms. In a third case the first symptoms were attributed by the patient to a supper of pork and potatoes consumed a few hours before.

As regards the twenty-seven fatal cases, there is a further point of importance. In five of them, so soon as the abdominal pain set in, violent purgatives were given, in most cases by the mother. In one case castor-oil, salts, and senna were successively taken without result, but a chemist who was consulted prepared a draught which had the desired effect. It is at least possible that these cases might have had a happier ending under a less active parent.

Symptoms. It is not necessary to describe the symptoms in these patients at any length. It is from such cases that the classical descriptions of peritonitis have been written.

The patient is suddenly attacked with pain in the abdomen, which is, so far as I know, never localised in the right iliac fossa. It is usually so severe from the very first that the patient lies down or takes to his bed. He commonly vomits within a few minutes or hours of the onset of the pain, and vomiting may continue throughout the illness. The temperature is raised from the very first, but seldom beyond 102 or 103. The pulse is greatly quickened. The mouth becomes dry, and the tongue furred. The urine is scanty, and often albuminous. The abdomen is from the first tender: in the course of twenty-four hours it becomes distended, and soon afterwards motionless.

In four of these thirty-six cases, where no purgative was given, two or three actions of the bowels accompanied the onset of the illness, and in two of these the bowels continued loose throughout.

Two patients were admitted on the second day of the illness, nine on the third day, eight on the fifth day, four on the sixth day, three on the seventh day, one on the eighth day, and four on the ninth day.

So that by the time the patient comes under hospital-treatment the disease is firmly established. The abdomen is much distended, absolutely motionless, and acutely tender. Veins can be sometimes seen running upwards from the groins. The pain remains severe, unless he is under the influence of morphia. He lies on his back with his knees drawn up: the eyes are sunken, and the face pinched and worn: the tongue and mouth are dry, and thirst is distressing.

The temperature commonly falls during the progress of the case, and may be normal or subnormal for the two or three days before the end. In some cases, however, death is preceded by a rise in the temperature to 104, 105, or 106. The pulse grows quicker and feebler. The hands become cold and the forehead moist, while the early anxiety and watchfulness pass into apathy, oblivion, and death.

In exceptional cases a wild delirium precedes the fatal issue, and it may be necessary to restrain the patient by force.

Not uncommonly, especially where the illness runs a long course, some fluid can be detected either in the flanks or above the symphysis pubis.

The fatal and the non-fatal cases run the same course and present the same appearance up to a certain point.

In the twenty-seven fatal cases death occurred in four on the fifth day, in three on the sixth day, in seven on the seventh day, in one on the eighth day, in three on the ninth day, in two on the tenth day, and the remaining cases proved fatal from the fourteenth to the nineteenth day.

On the other hand, in the nine non-fatal cases the general abdominal distension and tenderness passed away sufficiently to allow of the detection of a local hardening in the right iliac fossa. This point was reached in one case on the fifth day, in two on the sixth day, in one on the eighth, ninth, tenth, and twelfth days respectively, and in the remaining cases at later periods up to the end of the third week. Up to this point no man could say if the patient would live or die.

Gradual onset.

It has been already mentioned that in four of the cases the onset was a gradual one. All of these died. It is probable that these cases were at their start of the local form, and that a secondary infection of the general peritoneum took place, in consequence of the movements of the patient. They are of sufficient interest to be recorded.

CASE XI

Necrosis of tip of appendix: slow onset of symptoms: death by general peritonitis.

T. B., a man aged 32, was seized with slight pain in the abdomen while at work. He spent the greater part of the next day in bed, and took two aperient pills. For the next two days he managed to keep about, though the pain continued. On the fifth day of his illness the pain suddenly increased in severity and vomiting set in, and he applied for admission into hospital. He was found to be suffering from general peritonitis, but there was also some fulness and sense of resistance in the right iliac fossa. His extremities were cold and clammy, his pulse was feeble and rapid, and his temperature was 95·8. An operation was thought inadvisable, and he died four days later, on the ninth day of the illness.

At the post-mortem examination it was found that the tip of the appendix had sloughed and was perforated. There was general peritonitis, but this was most intense in the right iliac fossa and in the pelvis, while a collection of pus lay between bladder and rectum.

CASE XII

Concretion in and perforation of appendix: death by general peritonitis.

D. M., a lad aged 18, was seized with sudden pain and vomiting while engaged in washing milk-cans. On the next day he was rather better, and actually managed to do his day's work. On the third day, though the pain was still present, he again did his work, and in the evening he got a "hot draught" from a chemist and also took some castor-oil. He continued his work for three more days notwithstanding the pain, but in the evening of the third day (the sixth day of the illness) the abdominal pain suddenly became so severe that he fell on the ground and writhed. The next day he actually walked up to the hospital, and I found him sitting among the applicants for an out-patient letter, a sturdy stiff-necked lad with blue lips, with great beads of sweat on his forehead, cold clammy hands, and a feeble running pulse.

Without any delay the abdomen was opened, and a localised collection of pus evacuated from the right iliac fossa, but he was past hope.

It was found post-mortem that the appendix was nearly torn through about its mid-length, and outside it lay a laminated fæcal concretion. There was general peritonitis, with signs of a definite abscess-cavity in the iliac region.

It seems clear that the case began as a local suppurative peritonitis, and that infection of the general peritoneum occurred on the sixth day of illness.

CASE XIII

Ulceration and perforation of a cystic appendix: peritonitis at first local, afterwards general.

E. W., a woman aged 28, had had three previous attacks of peritonitis. In the first of these the peritonitis was general, and she narrowly escaped death: as the attack passed off an induration could be felt in the right groin.

In her present illness she was suddenly seized with pain all over the abdomen, but was not sick. She knew at once that it was the commencement of an attack similar to those which she had previously experienced, and she knew also that her last two attacks seemed to show a diminishing severity. Consequently she continued her daily work, though in constant pain and feeling ill throughout, for eight days. On the eighth day there was a sudden aggravation of the pain, constant vomiting set in, and she took to her bed, and died five days later.

It was found that the appendix had been a cystic one, but that it was now ulcerated, necrosed, and perforated in two places, and consequently collapsed. There was general hyperæmia of the peritoneum, but the signs of actual inflammation were limited to the lower half of the abdomen, and were most intense in the right iliac region and pelvis, where among the adherent intestines lay numerous pockets of pus.

CASE XIV

Necrosis of tip of appendix : slight illness for nine days : death by general peritonitis.

P. L., a boy aged 6, was taken exceedingly ill with abdominal pain and vomiting. The pain was at first general, but became localised in the right iliac fossa in two or three days. The illness ran the course of a simple perityphlitis for nine days, all the symptoms steadily abating in the usual way. On the ninth day there was a sudden return of severe pain and vomiting, and he was admitted into hospital, obviously suffering from general peritonitis. He lived through ten more days.

The tip of the appendix was found to be missing, and the end of the organ was black and soft. No concretion was discovered. There was general purulent peritonitis, but, as in the preceding cases, there was clear evidence of the formation of a definite abscess-cavity in the right iliac region.

It will be noted that in all these cases there was evidence, both in the history and in the post-mortem conditions, that the inflammation was at first local, and that the general peritonitis was a later development. The occurrence of cases such as these with a mild onset and the supervention of acute symptoms at a late period is a matter of great importance. They form a fairly large proportion of our cases of general peritonitis (four in thirty-six), and it is by no means improbable that, if they had from the first been placed under skilled treatment, the inflammation might have been limited to the region of the appendix, and they might have recovered.

Sudden collapse and death.

Another important point is the suddenness with which in any case of general peritonitis the patient may exhibit signs of collapse, and may die while the physician is debating within his mind the question of the advisability of operation.

Instances of this sudden change could be given from our cases, but a more marked example has been clearly recorded by Lees.[78] The patient, a man aged 27, complained one day of a general feeling of illness. Early the next day pain gradually appeared in the right iliac fossa, and he vomited two or three times. On the third day he was admitted into hospital. He had then slight fever, but pulse and respiration were normal. He did not look anxious. The abdomen was a little distended, the recti were rigid, respiration was entirely thoracic. He lay on his back, but the thighs were not flexed. He complained of a little abdominal pain, and there was moderate tenderness over the appendix.

So that it appeared to be a case of disease of the appendix with subacute peritonitis. "Yet within twenty-four hours he was in a hopeless condition," and he died in less than four days after the first onset of the pain, death being preceded (as is sometimes the case in peritonitis and intestinal obstruction) by the vomiting of dark fluid suggestive of altered blood.

It was found at the post-mortem examination that there was peritonitis over the greater part of the right side of the abdomen, and the cause of this was a perforation of the tip of the appendix, in which lay several earthy concretions.

I would put it that cases such as this, where a comparatively small portion of the peritoneal surface is inflamed and yet rapid death occurs, can only be explained on the theory of a bacterial origin of the peritonitis. When one speaks of "peritonitis" one is apt to picture merely the microscopical changes associated with the inflammation of a serous membrane—viz. the vascular phenomena, and the various forms of exudation. If peritonitis, as we know it clinically, consisted solely in these vascular and tissue-changes, it is not likely that it would be anything more than a local disease of an unimportant structure, presenting no grave symptoms and having no tendency to death. In actual fact, it is one of the most fatal conditions with which we have to deal. It is not likely that death occurs through the loss of the normal function of the peritoneum, for we may see a patient live for many months with his whole peritoneum converted into a dense fibrous structure. The fever cannot be the cause of death, for the rise in the body-temperature in acute peritonitis is too slight in itself to produce anything more than discomfort.

On the other hand, the peritoneal sac forms a suitable field for the growth of bacteria, and the serous surface responds to the introduction of bacteria by pouring out the best medium for their culture that we know.

The conclusion is forced on us that from the first day of an acute peritonitis, till recovery or death steps in, there is streaming through the vast absorbent system of the peritoneum some product of bacterial life which is always noxious and often deadly, now scanty in amount and now abundant. The poison may be resisted for many days or may be successfully excreted, but it may at any moment flood the system so that the patient drops back exhausted.

Treatment adopted

All these thirty-six cases were put more or less under the influence of opium. In eleven cases the abdomen was opened, the pus and inflammatory products within reach of the incision were removed, and an attempt made to wash out the whole abdominal cavity. All of these died, most of them within twenty-four hours, though one patient lived for nearly five days, but died eventually with the usual toxæmic symptoms. In three of these eleven cases the diseased appendix was removed at the operation.

Complications.

In one of the fatal cases of acute purulent peritonitis there was found post-mortem to be an empyema on the left side and commencing pericarditis. In another case one of numerous loculi of pus in the peritoneal cavity lay under the diaphragm on the right side. At this spot the diaphragm was perforated, and suppurative inflammation of the pleura had been set up. A similar case is mentioned by Johnston.[80]

In two of the nine cases which recovered, undoubted pleuritic friction was heard, in both cases on the right side.

RELAPSING PERITYPHLITIS

It is common to speak of a certain number of cases of perityphlitis as being of the "relapsing" or "recurrent" form. There can be no harm in this, if it is remembered that every individual who has once had an attack of perityphlitis is liable to subsequent attacks, so long as his appendix remains unobliterated or until it is removed by the surgeon. But it is in no sense a special form of the disease.

In many instances the attacks are limited to one or two in the year, and the period of liability to attacks extends to three or four years. But in the most severe form of the disease, to which the term "relapsing" is particularly applicable, the interval of comparative health between the attacks does not amount to more than two or three weeks, and the total result is then a nearly continuous illness, which may keep a man from his work for six months or more.

The question of surgical interference in these cases is one which presses for settlement. And it is necessary, before it is discussed, that such facts as we have relative to the occurrence and causation of these recurrent attacks should be clearly put forward.

If the origin of these attacks lies in the appendix, it is clear that an attack may occur at any time in any individual, whose appendix is in one of the morbid conditions previously described. Some of these conditions tend to a complete fibrous obliteration of the lumen, and it may be accepted that all danger then ceases. In some, on the other hand, such obliteration is necessarily impossible.

It is also clearly proved that the appendix may remain in a diseased and dangerous condition for some years, and yet only once or twice may there be an outbreak of peritoneal inflammation around it.

Frequency of recurrence.

It is of the first importance that we should know with some degree of certainty the frequency with which a recurrence of attacks is met with.

I have found that it is impossible to gain certain information on this point, because our patients cannot be followed up for any length of time. If they remain long in the same neighbourhood, a recurrence of symptoms is sure to bring them back to St. Thomas's Hospital, but as often as not they shift their habitation every year or so, and naturally seek the nearest hospital when taken ill.

It follows that the figures, which we have, show only the "minimum" frequency of recurrence.

Now of 250 patients suffering from peritonitis, local or general, due to disease of the appendix, fifty-nine had had previous attacks, and in four others subsequent attacks occurred. There can be no question as to the accuracy of these figures so far as they go. They show that in 23·6 per cent., at any rate, more than one attack occurs.

These patients were all drawn from the working class, and it is difficult to grasp the immense waste of time entailed by this disease in these fifty-nine individuals. If put in tabular form, some idea of this may be gained. All these attacks preceded the one for which the patient was admitted.

In	1	case	5	attacks	occurred	in	the	preceding	8	years.
,,	1	,,	5	,,	,,	,,	,,	,,	6	months.
,,	1	,,	4	,,	,,	,,	,,	,,	$2\frac{1}{2}$	years.
,,	1	,,	4	,,	,,	,,	,,	,,	2	,,
,,	4	,,	4	,,	,,	,,	,,	,,	1	year.
,,	1	,,	3	,,	,,	,,	,,	,,	8	years.
,,	1	,,	3	,,	,,	,,	,,	,,	7	,,
,,	2	,,	3	,,	,,	,,	,,	,,	2	,,
,,	3	,,	3	,,	,,	,,	,,	,,	9	months
,,	1	,,	2	,,	,,	,,	,,	,,	4	years
,,	1	,,	2	,,	,,	,,	,,	,,	3	,,
,,	1	,,	2	,,	,,	,,	,,	,,	2	,,
,,	3	,,	2	,,	,,	,,	,,	,,	1	year.
,,	3	,,	2	,,	,,	,,	,,	,,	6	months
,,	3	,,	1	,,	,,	,,	,,	,,	5	years
,,	2	,,	1	,,	,,	,,	,,	,,	4	,,
,,	1	,,	1	,,	,,	,,	,,	,,	3	,,
,,	1	,,	1	,,	,,	,,	,,	,,	2	,,
,,	6	,,	1	,,	,,	,,	,,	,,	1	year.
,,	8	,,	1	,,	,,	,,	,,	,,	6	months.
,,	8	,,	1	,,	,,	less than 6 months before.				

This table gives us an idea as to the period over which attacks of perityphlitis may be spread: the maximum of eight years has been exceeded in rare cases elsewhere recorded. In some cases also the number of definite attacks has been enormous: as many as fifty attacks spread over eight years have fallen to the share of one individual. Twelve attacks have been known to occur in one year.

But this table obviously does not contain the whole truth. We ought to know how many of these 250 patients had subsequent attacks after leaving the hospital, and this information cannot be obtained on any large scale, because they cannot be traced beyond a period of eighteen months or two years.

Yet, taking this limited period only, we find many instances of subsequent attacks, but the actual figures are too small to be of any value.

So that we must rest content with knowing that the minimum frequency of recurrence is as follows:—

In 23·6 per cent. more than 1 attack.
,, 9·6 ,, ,, 2 attacks.
,, 6·0 ,, ,, 3 ,,
,, 3·2 ,, ,, 4 ,,
,, ·8 ,, ,, 5 ,,

In his original paper in 1886 Fitz calculated that recurrence occurred in 11 per cent. Subsequent experience[79] has led him to place it as high as 44 per cent.

Krafft states that of 106 patients 23 per cent. had had previous attacks, a figure closely agreeing with my own.

The comparative rarity of previous attacks in cases of acute general peritonitis has been already mentioned.

In two cases only, so far as can be ascertained, did any attack follow the local suppurative form of the disease, and in one of these cases a local abscess was again formed. Indeed, there is evidence to show that a perityphlitic abscess is in some measure protective against future trouble of any kind in the region of the appendix, probably through the density and strength of the adhesions formed.

Morbid conditions of the appendix.

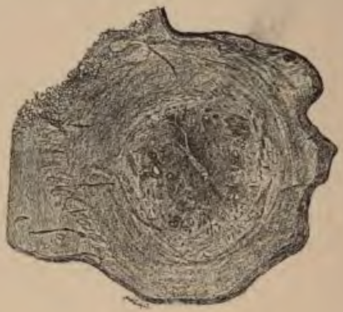
Inasmuch as the attacks in these recurrent cases are usually of the simple perityphlitic form without suppuration, we should expect that the conditions of the appendix would be much the same as those which have been mentioned as being the basis of a simple perityphlitis. But we should expect that the conditions would be such as would militate against obliteration of the appendix.

Such figures as we have bear out these expectations, though they are necessarily not on a large scale, for these cases are seldom fatal. Of fourteen cases derived from various sources, in six there was a cystic appendix (in two due to kinking), in five there was a chronic catarrh with thickening of the appendix-wall, in two a concretion was present without perforation, and in one there was an infective appendicitis.

So that we may say that the condition of the appendix which is, above all others, associated with a relapsing form of local peritonitis is a chronic catarrh, in which either the wall of the tube is enormously thickened, or obliteration of the lumen has occurred at some point with a cystic distension on the distal side. A good instance where both these results occurred to some extent is shown in Figs. 21 and 22.

The appendix here represented was excised by Mr. Clutton from a patient under my care, who had had four attacks of simple perityphlitis in three months. The operation was performed at the time which I believe to be the most favourable moment for interference—viz. in this particular instance seven days after the complete subsidence of all signs of inflammation in the right iliac fossa.

The appendix was removed with great ease. It proved to be three inches long, lying under the cæcum and rather to its outer side, and loosely adherent. It was considerably enlarged, the average outside transverse diameter in the fresh state being $\frac{2}{5}$ of an inch. It was as hard as a man's little finger.



× 5.

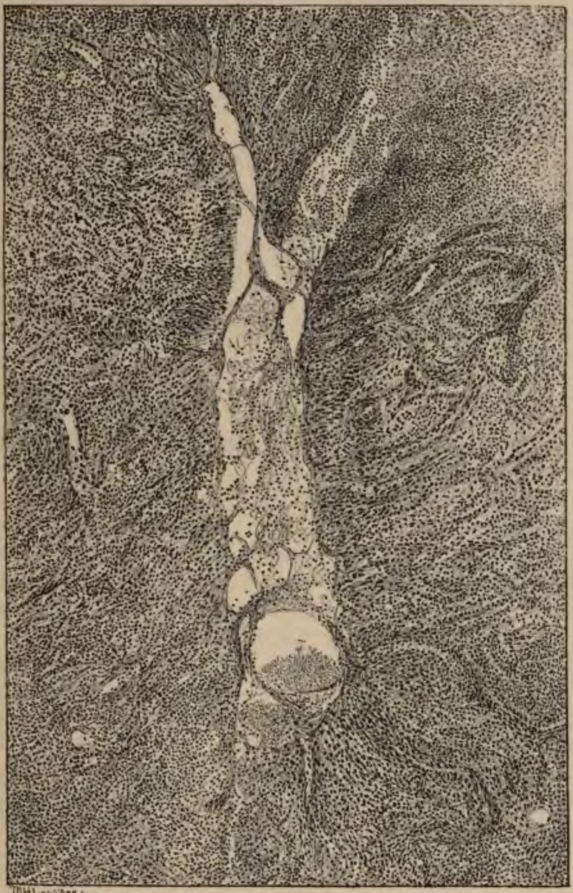
Fig. 21

Transverse section of appendix, taken near its cæcal end, showing the great thickening of the wall due to chronic inflammation and the nearly complete obliteration of the lumen.

Now it is seen by microscopical examination that the enlargement is due to the thickening of all the structures composing its wall, peritoneum, muscular coats, submucosa, and mucosa. But its hardness is due to the fact that the organ is nearly solid. The lumen is reduced to a narrow slit, the relation of which to the wall-thickness is shown by Fig. 21.

The mucous lining, as such, has in fact ceased to exist. In its deeper part there is still a considerable amount of lymphatic tissue, but it contains also a large basis of newly formed fibrous tissue. There is no trace of epithelial lining to the lumen, and no remnant of a crypt can be seen. The inner half of the original mucosa is composed of pure fibrifying granulation-tissue, as is shown in Fig. 22.

Here then is a perfect example of a chronic appendicitis, leading to great thickening of the tube, such as is commonly found as the cause of relapsing perityphlitis. The process of obliteration of the lumen is evidently making active progress, and is as a matter of fact not far from completion, and if this process of obliteration had been taking place to an equal extent throughout the whole length of the appendix, it would in a short time have ceased to be a source of disease.



× 75.

Fig. 22.

The lumen of the appendix shown in Fig. 21, more highly magnified. Epithelium, crypts of Lieberkühn, and basement-membrane have all disappeared: the narrow slit which represents the lumen contains some inflammatory exudation and recently shed blood, and it is bounded on all sides by granulation-tissue and fully formed fibrous tissue.

But in this specimen, as is commonly the case, obliteration is further advanced in the cæcal part of the tube than in the extremity. Moreover, towards the extremity of the tube there are still remnants of epithelium to be found. So that, although a natural cure in this case was almost in sight, there still remained a great danger in the obliteration of the cæcal part of the lumen with subsequent distension of the distal part by a purulent or mucous fluid.

Symptoms. As regards the symptoms in cases of the recurrent form, not a word need be added to the description of simple perityphlitis already added. Each attack is complete in itself and runs the same course, though there is a tendency towards a diminution of severity as time goes on.

Between the attacks, moreover, the patient's condition is not always a happy one. Morbid sensations not uncommonly hover round the appendix. Many patients complain of a sensation of "weakness" at that spot; they grow shy of exercise, and learn to shudder at the thought of constipation and the necessary purgative.

It is matter for wonder that an "appendicular hypochondriasis" is not a common occurrence. Fortunately the general public are less acquainted with the appendix than with the rectum and œsophagus.

Result. There can be no doubt that the great majority of these cases attain an ultimate cure. The mortality is extremely small, though no actual figures can be cited on this point. The possibility of a general peritonitis probably diminishes with every attack, yet such an event may happen from rupture of a cyst, as in two cases already narrated in this paper.

The possibility also of subsequent fatal obstruction of the bowels must be borne in mind. Of the four cases of this kind previously detailed, in three the adhesions which caused death were the outcome of previous attacks.

The importance of this tendency to recurrence lies rather in waste of time than in waste of life, and in theory at any rate it is possible to prevent it.

CHAPTER VIII

Medical Treatment.—Simple perityphlitis, general peritonitis.
Surgical Treatment—(*a*) History of surgical treatment of perityphlitic abscess. Dupuytren, Grisolle, Battersby, Hancock, Willard Parker, Bull.
(*b*) History of preventive treatment by excision. Sands, Treves, Bull, McBurney, Senn, Price, Weir, Keen, Morris, Porter.
(*c*) Comparison of radical and conservative methods of treatment. The claims of the advocates of early excision.
(*d*) Conclusions.
(*e*) Surgical treatment of perityphlitic abscess, general peritonitis, and relapsing perityphlitis.

MEDICAL TREATMENT OF APPENDICULAR PERITONITIS

Simple perityphlitis.

THE part played by the physician in the treatment of a simple perityphlitis is not unimportant.

In the first place, it rests with him to decide in what cases a radical surgical cure must be attempted, and the grounds on which a decision must be founded will be subsequently put forward.

If the radical treatment, which will be subsequently described as being largely practised in America, is not fully accepted, it still remains the duty of the physician to watch closely in the early days of the illness for the formation of pus and for the supervention of a general peritonitis, and in either case the necessity for active measures can hardly be doubted.

So that there is demanded from the physician in the first place an accurate knowledge of the natural history of the disease.

For the rest, his care is directed to the relief of symptoms and to the avoidance of causes which might tend to convert a local peritonitis into the diffused form.

The pain is relieved either by warm or by cold applications, poultices, fomentations, or the icebag. The use of leeches over the affected part is in almost all cases followed by great abatement or complete cessation of the pain. An objection has been raised to their use on the ground of the difficulty of thoroughly cleansing their bites, if laparotomy should be subsequently required. But there can be no difficulty in so arranging the leeches as to avoid the usual line of incision.

Opium is an essential, preferably given by hypodermic injection. The objects of its use are to allay pain and to prevent peristaltic movement of the intestine lying in the right iliac fossa. The latter object is soon attained in nearly all cases, peristalsis being already hindered by the peritoneal inflammation. And the pain is quickly subdued by the local applications above mentioned. And although some opium is necessary, it is important not to continue its use when these two objects have been gained, and as a rule it is seldom required beyond the first two or three days.

Absolute rest in bed from the first onset of symptoms is an obvious necessity.

As regards the management of the bowels, it is best to err on the safe side of constipation. No purgative, saline or otherwise, should be given until the patient is well enough to leave his bed. A glycerine enema or a glycerine suppository acts without fail whenever required. The time at which an action of the bowels may be safely provoked will vary in different cases. As a rule it is safe from the fifth or sixth day onwards, but if the case exhibits severe local symptoms at this point it is better to wait.

Nothing beyond a fluid diet is required until the fever has subsided.

Finally, when the patient is able to leave his bed, his bowels do not at once resume a regular action. Aperient mineral waters may then be safely used, supplemented if necessary by glycerine suppositories.

And on resuming active life the light pressure of a pad over the right iliac region, kept in place by a belt, certainly relieves the sensations of uneasiness which are so commonly present. And such pressure is possibly of actual use in promoting obliteration of the appendix.

General peritonitis.

The medical treatment of a general peritonitis from appendix-disease presents no special points. It is confined to skilful feeding, the maintenance of strength, and the alleviation of pain.

The most important point is the sparing use of opium in this as in all conditions where an operation may become necessary. A patient suffering from peritonitis and under the influence of opium is seen as through a glass, darkly. If he is thoroughly under its influence, the effect is so great that it may be difficult to take a serious view of the case. It is not possible to make that correct estimate of the vital condition which is of such great importance in deciding as to the advisability of operation, and surgical relief may be postponed in consequence until the final collapse has set in.

SURGICAL TREATMENT OF APPENDICULAR PERITONITIS

The various opinions as to the necessity of surgical treatment of the different forms of peritonitis due to disease of the appendix present a bewildering want of agreement. If the medical literature of each country be taken as a reflection of its current opinion, a wide division becomes apparent, and even at the discussions instituted at medical societies there is exhibited a surprising difference of opinion among the members.

This is in part due to the want of uniformity in nomenclature, so that one man knows not to what class of case his fellow alludes. But the main cause of the discrepancy is the protean nature of the disease. One man " has never had a fatal case," and cannot believe that surgical treatment is ever permissible. Another finds a heavy mortality, and thinks that many lives might have been saved by timely operation. While a third has already operated on a large number of cases and removed the appendix with a mortality of two per cent., but if pressed by inquiry he would find it difficult to forecast the result, if his patients had been left alone.

In order to understand clearly the present position, it is well to trace the progress of opinion upwards through the last fifty years.

History of the surgical treatment of peri-typhlitic abscess.

As might be expected, the form of appendicular peritonitis which first fell into the hands of the surgeon was that which results in a perityphlitic abscess.

Little if any active treatment was employed in the first thirty years of this century. Dupuytren (*loc. cit.*), for example, in 1833 recommended the use of purgatives in order to hasten the evacuation of the iliac abscess into the bowel.

Grisolle (*loc. cit.*), on the other hand, maintained in 1839 that an abscess in this position must be incised and not allowed to discharge either externally or into the bowel.

Battersby[81] in 1847 recommended early evacuation. Hancock[82] a year later expressed the opinion that an incision should in some cases be made even before fluctuation appeared, and he published a case of the kind where an operation was quite successful.

It is quite clear, however, that for some years after this it was a common practice to allow a perityphlitic abscess to discharge into the bowel or through the abdominal wall.

Willard Parker[83] in 1867 went a step further than his predecessors, in insisting on an early operation in cases of localised appendicular peritonitis. He himself operated on three cases successfully. In his view it was not wise to explore the inflammatory mass before the fifth day of illness. He set apart these five days for the formation of adhesions, which should separate the inflammatory focus from the general peritoneal cavity. After the expiration of this time he was prepared to explore in all cases of doubt. If an abscess was in process of formation, the incision would tend at any rate to make it point in a safe direction; and, if no abscess should form, the incision would relieve tension, and would in itself do no harm.

Even so late as the year 1873 the opening of a perityphlitic abscess was not an established routine practice. And the explanation of this can be gathered from the course of the disease as shown by the statistics given in this paper. It will be seen that of our 228 cases, which presented the same onset of symptoms, no less than 190 recovered under simple medical treatment. So that, as all these cases were at that time placed entirely in the physician's hands, one can easily appreciate the temptation to wait for absorption, and to wait moreover hoping against hope, until the abscess showed through the abdominal wall or burst into the bowel.

The thesis written by Bull [84] in 1873 emphasised the view of Parker. It had the additional merit of recognising that in a large majority of cases of localised appendicular peritonitis recovery by resolution occurs. The treatment recommended by him deserves attention, for (with the exception of some minor details) it represents the practice generally adopted in this country at the present time. Given a patient in whom symptoms of localised peritonitis in the right iliac region make their appearance, the first step to be taken in his opinion was an attempt to promote resolution by absorption. For this end he recommended leeches, followed by warm fomentations, cold compresses, the use of opium, and the avoidance of purgatives. "This plan should be pursued only so long as all signs of suppuration are absent." He summarised the signs of suppuration as being "the first appearance of increase in size of the tumour, or augmentation of its pressure-effects, or increase in the fever with chills or sweats." As soon as suppuration was recognised, he was prepared to incise the tumour at once.

This brings us to the practice commonly adopted in London hospitals at the present date. In every case of localised peritonitis in the right iliac region a watch is kept for indication of the formation of pus, and so soon as this is recognised the inflammatory tumour is incised. The time at which the formation of pus is recognised varies greatly in the hands of different men, and it is even now not uncommon to admit into hospital a patient with a chronic abscess in this region having a capacity of half a pint or more.

Grisolle, Hancock, Parker, and Bull may be taken to advocate an operation for a perityphlitic abscess which should be too early rather than too late. The reasons advanced by them were the long and dangerous illness which followed the spontaneous rupture of an abscess, and the danger of pyæmia. But there was not at this time any ulterior motive aiming at the prevention of future attacks of the disease or at the prevention of a general peritonitis.

History of the preventive treatment by excision of the appendix.

About the year 1882 the theory that the appendix and not the cæcum was primarily at fault in these cases began to attract notice, and a few years later (after the appearance of the monograph of Fitz) it began to gain general credence.

By this time the old danger of opening the peritoneal cavity had been dispelled, and a great impetus had been given to all branches of abdominal surgery. From all quarters there were appearing accounts of attempts to deal radically with abdominal conditions, which had hitherto reposed in the hands of the physician.

So that at this moment there presented itself to thinking minds the conception of the appendix as an organ, which, on the one hand, is suited to complete removal by reason of its shape, arrangement, and general uselessness, and on the other hand, is liable to disease from which a fatal peritonitis frequently ensues. Hence arose in some quarters the practice of routine excision of the appendix so soon as the presence of disease in it could be recognised.

Now whether this practice is intrinsically right or not, whether its adoption is a step on firm ground or a step from which retreat must be effected, it must be admitted that it was founded to some extent on false bases, and on an imperfect knowledge of the disease. The same may be said of many notable advances in methods of treatment, and this criticism contains no reflection on the American surgeons with whom the practice originated.

In the first place, the idea of an early excision of the appendix was initiated at a time when the results of disease of that organ were still generally thought to be extra-peritoneal, and the actual founder of this method of treatment held this belief in its entirety. This misconception was speedily and completely removed, and it has no further bearing on the question at issue.

In the next place, I think there can be no doubt that excision of the appendix was originally brought forward under an exaggerated idea of the gravity and fatality of appendicular peritonitis. The rapidly fatal form of general peritonitis is an impressive spectacle, and it gave a colouring to the whole conception of the disease. The gravity of disease of the appendix was measured by the rapidity of death in a few fatal cases rather than by the actual frequency with which a fatal event occurs.

This error arose from the fact that even at that time the cæcum was a prominent factor, and the great bulk of the cases of non-suppurative perityphlitis which occur in the practice of physicians were still vaguely connected with the old idea of fæcal impaction in the cæcum.

In other words, the preventive method of treatment by early removal of the appendix was originally undertaken with the idea that by its means a large number of patients were to be saved either from death by general peritonitis or from the long illness associated with a perityphlitic abscess. The two severe forms of peritonitis were accepted as the type of disease to be combated, rather than the far more common form of simple perityphlitis which has no tendency to death, and which need cause no anxiety to patient or physician.

Owing to the lack of any trustworthy statistics as to the absolute mortality arising from disease of the appendix, the same high estimate of its gravity can be read in all subsequent writings on this subject.

The preventive plan of treatment was initiated by Sands[85] in 1887, and he was the first man to excise the appendix successfully after its perforation.

In the same year Treves[86] advised excision as the rational method of dealing with cases of relapsing perityphlitis.

In 1888 Bull[87] explained his views before the Medical Society of London. They were essentially conservative, and (with the exception of the use of the exploring-needle) they obtained a very general agreement in this country. He recognises three forms of perityphlitis—viz. "a resolving and suppurative form, each marked by definite symptoms in some cases, in others difficult to distinguish; the suppurative form, again, may be spreading or circumscribed." This is practically the classification that has been used throughout this paper. Considering these three forms in detail, he decides to leave the "resolving perityphlitis" to nature, to evacuate the pus of a "perityphlitic abscess" by the end of the first week of illness, and to perform laparotomy at once so soon as a diagnosis of a spreading peritonitis can be established.

So far his view is clear enough, and it is generally commended. But at this point he is brought face to face with the practical difficulties attending the early recognition both of the presence of pus and of the existence of a spreading or general peritonitis. It is easy to make the rule to incise a perityphlitic abscess at the end of the first week, and to perform laparotomy directly a general peritonitis is recognised. But by the symptoms alone it is not always possible to detect the presence of pus so early as the end of the first week, and he consequently recommends exploration with a needle in such cases. And further, the difficulty of recognising a spreading general peritonitis in the first twenty-four or forty-eight hours is equally great, so that in cases of doubt he would recommend early exploration, for "the risk of exploration is less than the risk of the disease."

As regards the propriety of removal of the appendix in recurrent cases he is undecided, being of opinion that a certain number of such cases are due to disease of the cæcum.

McBurney's[88] position appears at first sight to be of a radical nature. It is, however, in a manner the logical conclusion which results from a free confession that at an early period (the first twenty-four or forty-eight hours) we are unable to forecast the progress of the case. A patient is suddenly taken ill with a definite series of symptoms, which at once suggest the onset of some degree of peritonitis round a diseased appendix. I believe myself that in the majority of such cases no man in the first twenty-four hours can even guess as to whether the appendix is perforated or necrosed and a general peritonitis has begun, or whether it is merely the seat of a less acute process from which a simple adhesive peritonitis or a local perityphlitic abscess will be the outcome. Excepting some cases of very acute spreading peritonitis, I believe this confession is unavoidable.

McBurney makes this confession of ignorance, and his words are worth quoting. "The only real difficulty lies in determining within the first few hours what the future progress of the disease is to be, in deciding whether firm adhesions are forming which will effectually exclude pus from the general peritoneal cavity and so provide for subsequent safe evacuation of the abscess, or whether no such protecting wall exists, and an over-distended appendix threatens to instantly set up a fatal peritonitis. . . . The pathological conditions of the appendix, as compared with the symptoms in my own cases, most positively show that one cannot with accuracy determine from the symptoms the extent and severity of the disease. . . . If this difficulty could be set aside by a more careful study of symptoms, and without losing valuable time, our course would be clear, and we should no longer helplessly hesitate as to when to operate and when to stand aside. There is no reason to think, however, that diagnosis from symptoms alone will ever reach that perfection. We need some further aid to diagnosis."

Now this "further aid" in McBurney's opinion can only come through an exploratory incision and direct inspection of the appendix. Having reached this point, the only conclusion is that the rational treatment of incipient appendicular peritonitis is removal of the appendix as soon as possible after the first onset of symptoms. "If," he says, "it can be shown by future experience, with improved methods of operation and with more perfect antiseptic precautions, that the exploratory incision for the inspection of the diseased appendix is much more free from danger than the expectant treatment, then there could be but one answer to the question, "What is the best treatment?"

Senn's[89] views, as published in 1889, tend in the same direction. When disease of the appendix is recognised, it must be extirpated, lest perforation occur. "It may be stated as a general rule, to which there can be but few exceptions, that the appendix should be extirpated in all cases where from the symptoms and history of the case the existence of a localised destructive inflammatory process can be surmised. . . . Extirpation of the appendix at a time before the inflammatory process has reached the serous coat is one of the easiest and safest of all abdominal operations. . . . All cases of catarrhal and ulcerative appendicitis should be treated by laparotomy and excision of the appendix as soon as the lesion can be recognised."

In a second paper published in 1891 McBurney[88] reiterates his views, basing his recommendations of an early operation on the impossibility of forecasting the course which any individual case will take. "Out of a number of cases all beginning in a somewhat similar manner, supposing them all to have been treated conservatively, at the end of a week some will have died from general peritonitis, others will be well or on the road to recovery, and others again will be waiting for the surgeon to open the abscess, running the risk from hour to hour of serious accident."

He appends a table of twenty-four cases in which he operated at the earliest possible moment. The appendix was completely removed in all cases but two; in one, partial removal was deemed sufficient; and in another, removal was deemed unwise. Recovery occurred in every case except one, and in this case it does not appear that the operation in any way contributed to death.

The earliest time of operation was the twenty-fourth hour of the illness, one was performed at the fortieth hour, seven about the end of the second day, nine on the third day, two on the fourth day, two at the end of the first week.

The views expressed by Senn and McBurney probably represent the current of thought in America at the present time. Numerous contributions to this subject have appeared in the last three years, and all of these tend in the same radical direction as regards treatment, though they differ to some extent on points of pathology.

Price[90] argues in favour of exploration in all cases without waiting for a positive diagnosis.

Weir[91] writes mainly of the chronic relapsing form, and his opinion, which is the result of mature reflection, is characterised by great moderation. He would operate in the quiescent intervals of relapsing cases, when the patient is debarred from the enjoyment of life or the ability to earn a living.

Keen[92] is in favour of immediate operation in all cases of appendicular peritonitis, excepting only "the mild form of appendicitis without perforation, ending usually in resolution without the formation of an abscess." He stands alone in thinking that by any symptom or symptoms these "mild cases" can be distinguished at an early period from the more severe forms associated with perforation.

I would refer again to the opinion of McBurney, with which no one who is brought into frequent contact with the disease can fail to agree, that it is not possible, in the majority of cases, to distinguish between the various forms of appendix-disease and between the degrees of resulting peritonitis, except by watching the progress of the case, and we are wise only after the event. In other words, in the majority of instances all forms of peritonitis are commonly ushered in with the same train of symptoms.

Morris[93] holds to the consistent views of Senn and McBurney. "I am in favour of early operative treatment in practically all cases of appendicitis, in view of the fact that the inflammation is so infectious in character. . . . The simple diagnosis of appendicitis I hold to be sufficient excuse for operation, and as soon as this stand is taken by physicians generally, the patients who would die from the disease under procrastinating treatment will be spared to their families and friends, to say nothing of saving time and relieving anxiety for patients who have exacerbations."

Finally, Porter[94] has recently published some actual figures which are of great value, and may serve as a basis for a consideration of the exploratory and preventive method of treatment. His table is quoted in full.

Whole number of cases 448
do. recoveries 371
do. deaths 77
average mortality 17·23 per cent.
Of these 448 cases :—
(*a*) removal of appendix during attack—
recovered 122 = 80·3 per cent.
died 29 = 19·7 ,,
151
(*b*) removal of appendix during quiescence—
recovered 13 = 92·86 per cent.
died 1 = 7·14 ,,
14
(*c*) incision and drainage of abscess—
recovered 154 = 81·82 per cent.
died 34 = 18·18 ,,
188
(*d*) appendicitis without operation—
recovered 82 = 86·32 per cent.
died 13 = 13·62 ,,
95

It may be accepted, then, that there is at the present time in America a large volume of opinion in favour of operation and the removal of the appendix at the earliest possible moment after the onset of any peritonic symptoms, mild or severe.

A faint echo of this may be heard in Germany and France.

In our own country the radical plan has been seldom resorted to. In a large number of cases, after the recommendation of Treves, the appendix has been excised in the quiescent interval of relapsing cases, and in an unknown number of cases of general peritonitis the abdomen has been opened in the middle line, with the idea of grappling with the already established general infection, and in many of these the appendix has been removed. Of this class the published recoveries may be counted on the fingers of one hand.

The general plan of treatment hitherto adopted in this country is somewhat as follows. I do not think I am overstating its conservative character. Given a patient in whom the symptoms appear which have been already detailed as denoting the onset of appendicular peritonitis, he is dealt with after the fashion which was described by Bull in 1873 as being his own practice. An attempt is made to subdue the inflammation and to promote the absorption of the coagulable inflammatory products. A jealous watch is maintained for the formation of pus, and it is released by incision at the earliest possible moment, the appendix not being removed unless it obtrudes itself upon the surgeon's notice in the abscess-cavity. When it appears manifest that a general peritonitis is in action, the treatment varies in different hands, but as a rule the abdomen is opened and washed out, and, unless great difficulties offer, the appendix is at the same time removed.

Comparison of the radical and conservative methods of treatment.

It is possible that the American and the English methods of treatment, the radical and the conservative, will in a long series of observations be found to yield the same life-saving result. It is possible, however, that there is a great difference in the total death-roll associated with these widely divergent views. But it is clearly of importance that the question should be speedily settled, and a settlement can only be effected through the cold figures of past lives and deaths.

One rises from the perusal of the abundant American literature on this subject feeling nothing but admiration for the work of such men as Bull, McBurney, Senn, and others. But some of the more recent contributions to the purely surgical question appear to be open to criticism. It is a criticism applied to the arguments employed rather than to the value of the treatment which is urged. They seem to be compiled by a surgeon for the use of a physician, concerning a disease which is in all its aspects familiar to the physician, but of which the surgeon has seen only the more severe form. They assume a rate of mortality from disease of the appendix which has no foundation in actual figures, and which if it occurred in the practice of any one man would entitle him to nothing but compassion. The treatment advocated may, however be none the less correct.

The first requisite, in an attempt to decide on a definite line of treatment, must be a clear knowledge of the natural course of the disease in all its forms. I believe that this natural course can be learnt from the subjoined table of 264 cases which were admitted into St. Thomas's Hospital, in consecutive order. At any rate, this series of cases is certainly not open to objection on the score of undue surgical treatment.

	Deaths.	Recoveries.	Total.
a. Peritonitis limited to the right iliac region and not proceeding to the formation of pus (Perityphlitis)	0	190	190
b. Peritonitis similarly localised but ending in the formation of pus (Perityphlitic abscess) .	10	28	38
c. General Peritonitis	27	9	36
	37	227	264

The sum of the surgical treatment employed in these three classes is as follows:—

a. In one case of simple perityphlitis the appendix was excised during an attack.

b. In 33 cases a perityphlitic abscess was opened and drained. In none of these was an attempt made to excise the appendix.

c. In 11 cases of general peritonitis laparotomy and flushing was carried out, and all of them died. In three of these the appendix was removed during the operation.

I think we may safely conclude from these figures that, taking all forms of appendicular peritonitis together, the total mortality under the conservative method of treatment is at the rate of 14 per cent.

I think it is probable also that, without any radical alteration in the line of treatment, a similar series of cases in years to come will show a reduction in this rate of mortality to 12 per cent., or even less. This view is based on the fact that in two of the fatal cases of perityphlitic abscess the abdomen was opened in the middle line, and a general infection of the peritoneum was thus set up by breaking down the natural limiting adhesions. And in four of the twenty-seven fatal cases of general peritonitis the peritonitis was at first local, and subsequent general infection of the peritoneum occurred, through the refusal or inability of the patient to keep at rest before his admission into the hospital.

I believe it is only by examination of figures such as these that a true verdict can be given as between the radical and conservative methods of treatment.

Now it will be remembered that it is claimed by the advocates of the radical plan of excision of the appendix at the first onset of peritonitic symptoms—(1) that many lives will be saved and much suffering avoided by the prevention of future attacks; and (2) that by such early excision the attack of peritonitis which is beginning at the time of operation will be cut short, and that, as some of these cases if left alone terminate in general peritonitis, a number of lives will be thereby saved.

The first claim—the saving of life by the prevention of subsequent attacks.

It will be well to keep these two claims distinct and to consider them separately.

In deciding as to the first claim, we must learn what would have been the result in our cases if in all of them the appendix had been removed either during or immediately after the first attack. And in considering this we will make the large assumption that in all cases a complete and successful excision was possible, and that in no case did any accident occur in the operation by septic infection of the peritoneum or by any other means.

Now fifty-nine of our 264 patients had had one or more previous attacks, and these fifty-nine are distributed among our three classes in the following way:—

(*a*) Forty-five belong to the class of simple perityphlitis, and all of these recovered.

(*b*) Seven belong to the class of perityphlitic abscess, and of these seven patients four died and three recovered.

(*c*) Seven belong to the class of general peritonitis, and of these seven patients three died and four recovered.

So that, if we paint the picture of early excision of the appendix in the very brightest colours, we arrive at the following result. Of our 264 patients, forty-five would have been spared an attack of simple perityphlitis; seven more would have been spared the formation of a perityphlitic abscess, and of these four lives would have been saved which were lost; and seven more would have been spared an attack of general peritonitis, and of these three lives would have been saved.

In other words, by operation during or after the first attack seven lives would have been saved, and fifty-two other individuals would have been spared a more or less severe illness.

Though these results probably fall far short of the estimate formed by some of the advanced American school, they are nevertheless impressive. And they are moreover true, and up to this point involve nothing that is doubtful. But before a definite decision can be given as to the real merits of this plan of preventive treatment, an uncertain factor must be faced. It will be seen that in the hypothetical saving of these seven lives, and in the obviation of future illness in these fifty-two other patients, it will have been necessary to open the peritoneal cavity over the right iliac fossa and remove the appendix in fifty-nine cases without any mishap or accident.

It is open to every one to form his own opinion as to the possibility of such wholesale successful surgery. I believe that the best American operators regard the mortality attending removal of the appendix as being practically nil in such uncomplicated cases as we are considering.

At any rate, with proper care it could hardly amount to anything like seven deaths in fifty-nine cases (12 per cent.)

I believe that the figures here given present the matter in such a light that every one may draw a conclusion from them. For my own part, I consider that if the mortality attending the operation itself is, as appears probable, either nil or very small, then the claim as to the saving of some lives and the prevention of more subsequent illness must certainly be allowed.

The second claim—the cutting short of what might otherwise prove to be a fatal peritonitis.

In the second place, it is claimed that many lives would be saved by the routine practice of early excision (that is to say, excision in the first forty-eight hours of illness), inasmuch as a general peritonitis may be commencing which will prove fatal. This claim appears to be more doubtful than the other.

Now there are not, as a matter of hard fact, many lives that require to be saved in this way. It is clear that of every 100 cases only thirteen develop a general peritonitis, and of these not all die. But, even supposing that these thirteen must necessarily die unless the appendix is removed at the very outset, the claim still appears doubtful.

We are at once on uncertain ground. There are two elements of uncertainty.

In the first place, in order to save these thirteen lives, an operation involving the opening of the peritoneal cavity and the removal of the appendix must be carried out successfully and without accident in not fewer than 100 patients. Every one can form his own opinion as to the possibility of this. The mortality attending the operation may be extremely small, but still if it is a plus quantity it must be reckoned as a set-off to the theoretical benefit to be derived.

In the second place, is it certain that, if the appendix is successfully excised during the first two days of illness, a general peritonitis will be thereby prevented? A surgeon may excise twenty appendices in this way with complete success, and he may say that in so many of these cases he has prevented a general peritonitis. He is entitled to his opinion, but it can necessarily only be founded on the probability derived from the morbid conditions of the appendices removed.

Now I am myself inclined to doubt whether in the majority of the cases of general peritonitis, having a sudden onset, excision of the appendix, however early (unless perhaps in the first two or three hours), would be of any avail. In cases of sudden large perforation accompanied by the passage of fæcal matter and bacteria into the abdominal cavity, and in cases of rapid necrosis of the infective nature described in this paper, I believe that a bacterial peritonitis would, as a rule, run its fatal course unaltered.

It will be remembered, however, that some of the cases of fatal general peritonitis begin as a local disease, which becomes general after the lapse of several days, commonly through the attempts of the patient to continue at work. There is good reason to think that this class, where the general infection is preceded by a local peritonitis, might be saved if early excision of the appendix were practised; but in these particular instances the peritonitis would probably remain localised, if the patient were only kept at rest from the commencement of the illness. If such a patient will not keep at rest by reason of his own painful sensations and feeling of illness, he is not likely to do so for the purpose of submitting to an operation.

It is further probable that those cases (of which an example has been given) where a patient exhibits at first the symptoms only of a mild illness, but surprises the physician by suddenly becoming collapsed and dying on the fourth or fifth day, might by early excision be brought to a happier issue. But such cases are not of frequent occurrence.

Actual experience alone can settle the value of this claim, and the published cases (even in America) are not yet sufficient for this purpose. It is not fair to quote the one fatal case which occurred in McBurney's series (*loc. cit.*), because the patient had had a previous attack, and the resulting adhesions rendered complete excision impossible. Gerster,[95] however, records a case where there was no tumour palpable in the groin, but only local pain there, and the patient exhibited the symptoms of a deep general intoxication. Laparotomy was performed, and the appendix, which was totally gangrenous, removed; but the high temperature and septicæmic condition continued, and a fatal result ensued.

In conclusion, it appears probable that by a routine excision of the appendix at a very early period some few cases might be saved from a general peritonitis, but for my own part I doubt whether the resulting gain of life would on this plea alone be large enough to counterbalance the disadvantages of the wholesale surgery which such a practice would entail.

If, however, the mortality of the operation itself is so small that it may be neglected, and if I am wrong in doubting its efficacy in preventing a fatal general peritonitis, it must then be admitted that excision in the first two days of illness is the true and rational plan of treatment.

We must, then, look upon it as the true treatment in an ideal world peopled by perfect patients and perfect physicians and surgeons. But in this country it is none the less impossible on any large scale.

The whole kernel of this line of treatment lies in its "earliness." There are two golden days in which it may be carried out. Within twenty-four hours of the onset of the first symptoms it is probable that the mere operation of removal of the appendix in the hands of a skilled surgeon would in itself entail scarcely any risk, and it is just possible that a general peritonitis, if such be supervening, might in a few cases be prevented. After the lapse of forty-eight hours, however, every day adds to the difficulties of operation, while the possibility of preventing a general peritonitis vanishes rapidly. When three or four days have gone by, we have practically returned to the old-fashioned conservative method, and we must be content with favouring the subsidence of a simple perityphlitis (if the case turn out to be of that nature), or with watching patiently for the formation of a localised abscess, or with dealing with an established general peritonitis.

The essentials therefore in this method of treatment, directed at a prevention of the possible onset of a general peritonitis, are twofold. In the first place, the time of operation is limited to the first two days of illness, and, secondly, the surgeon must be on the spot having the requisite skill and experience in abdominal work. And I would ask, How often are both these essential conditions fulfilled?

In many cases the patient treats himself for several days, generally having recourse to purgatives. Any one who has lived in a hospital must have a vivid recollection of the number of patients who walk up and apply for admission with a large inflammatory mass already formed in the groin. Medical and surgical departments are sharply defined in this country, and the vast bulk of these cases are at first in the hands of men who necessarily cannot have the skill and experience of the hospital surgeon, and I would go so far as to say that it is rarely that the surgeon is brought into relation with the patient within the first four days of illness, except in cases of quickly established general peritonitis from perforation, which is the very condition which this method of treatment is intended to prevent.

So that, even if it be allowed in theory that early excision will do more good than harm by preventing the development of general peritonitis, there remains the fact that it is at present impracticable except in isolated instances. The adoption of such a practice can come only through the action of the general body of medical men, and not through hospital-example.

General conclusions

I believe, then, that the claim which rests on the prevention of an impending peritonitis cannot be held to be established. On the other hand, it appears to be proved that, if the appendix is successfully excised at the first attack, there will result the prevention of a large number of subsequent attacks and the saving of some lives which are lost in these subsequent attacks.

I think that this much must be admitted from the figures already given. At any rate, the figures are such that any one may form his own opinion on them.

Let a man put into the one scale the lives that will be saved and the amount of illness that will be obviated; into the other scale let him put what he considers to be a fair estimate of the risks entailed by this surgical treatment; and I would contend that the balance can only be read in one way.

Excision of the appendix after a first attack, as a better measure than excision during a first attack.

It is clear, however, that the same benefit will result from excision "after" the subsidence of the first attack as from excision "during" the first attack. If the idea of preventing a general peritonitis be given up, it makes no difference to the beneficial result, whether the operation be performed "during" a first attack or "after" it. And certainly it would be an advantage that the surgeon should deal with a quiescent rather than with an inflamed peritoneum. There is no longer any question of confining operation to the first two days of illness, and this plan of treatment consequently becomes perfectly practicable, even in this country.

Though this benefit to be derived by the prevention of subsequent attacks must be allowed in theory, it is still difficult to arrive at a definite practice. No half-measures are possible: the appendix must be excised in all cases or none, for no man by any known symptom can prophesy as to the occurrence of future attacks.

Now, on being placed at the bedside of a patient suffering from a first attack, one has to consider the prospects before him. It may be accepted that the odds against any subsequent attack are roughly as 3 to 1 (my own figures) or nearly as 5 to 2 (according to Fitz), and that, if he should have a subsequent attack, the odds against his dying in it are as 7 to 1.

The total chances against a fatal issue at any time are thus very great, and one may well hesitate to urge an operation upon the patient with the idea of providing against such a remote contingency.

But it is not merely a matter of life and death. It is a question of the future health and wage-earning capacity of young adults. It is true that even the most serious of relapsing cases do as a rule become cured in course of time, but the waste of time is immense.

If a man suffering from a first attack of perityphlitis knows well the chances for and against a recurrence of attacks, and if he is of the leisured class, I can conceive him weighing the possibilities, taking the risk in a speculative way, and declining operation. He is on a level with the man who, being troubled with a hernia, declines the offer of a radical cure.

Our way would be clear if we were able in any first attack to foretell whether this would also be the last attack, or whether it would be followed by a series of others spread over months or years. At present this is beyond our power, and I doubt if the study of symptoms alone will ever place it within our reach.

We are brought, then, to the logical conclusion that it is the duty of the physician to place the matter clearly before the patient, and to recommend to him the operation of removal of the appendix after a first attack of perityphlitis, on the ground of preventing the slight loss of life and the immense waste of time that is associated with a recurrence of attacks.

The only point remaining which can make me hesitate to accept this conclusion is one of which I am not qualified to speak. It is the question of subsequent yielding of the scar and the formation of a hernial protrusion. If this is likely to be a common and troublesome event, it must candidly be taken into consideration.

It is well known that such yielding does not occur in the scar after evacuation of a perityphlitic abscess, but the conditions under consideration are somewhat different, inasmuch as the operation here recommended is performed in the absence of acute inflammation of the peritoneum in the region affected, and the wound is likely to heal with great rapidity.

The American surgeons seldom allude to it, but their experience must now be wide enough to justify a definite statement on this point.

In one case under my care where this operation was practised, at the end of four years the scar has certainly yielded to some extent, but it in no way inconveniences the patient.

Finally, as to the exact time at which removal of the appendix is best carried out, a surgeon accustomed to deal with the peritoneum is the best judge. Treves is in favour of waiting for complete subsidence of all peritoneal inflammation. So far as my experience goes, the most favourable opportunity is the time when all inflammation is at end, and yet such adhesions as may be in process of formation have not acquired strength and density.

The surgical treatment of the various forms that actually present themselves.

As regards the treatment which must be adopted in the various cases, as they actually come before us at the present time, there is not much to be added.

The signs by which suppuration may be recognised have been already detailed, and no one, be he physician or surgeon, would hesitate to empty the abscess-cavity by an incision over the tumour at the earliest possible moment.

(a) Perityphlitic abscess.

As regards removal of the appendix after evacuation of the abscess, I am convinced that it is not wise to make any close search after it. A perityphlitic abscess is almost a cure in itself, and it is very rarely that any subsequent trouble ensues. And as a general rule the diseased organ is so concealed by inflammatory exudation that an over-active search must endanger the limiting adhesions upon which the patient's life depends.

A perityphlitic abscess occurs at the rate of fourteen in a hundred cases of all forms of appendicular peritonitis.

(b) General peritonitis.

Though nine out of our thirty-six patients, who developed general peritonitis, recovered without operation, I do not believe that this is any justification for not washing out the abdominal cavity and removing the appendix in any case where it can be undertaken with any chance of success. Our experience at St. Thomas's Hospital is one of constant failure, and I believe the same story is heard from all London hospitals.

The duration of the illness before admission into hospital has been already given in thirty-one instances, and most of these were past hope when admitted. Some few cases of successful operation early in the height of general peritonitis have been recorded both in this country and in America. They are sufficient to assure us that a better general result might be obtained if they came under hospital treatment at an earlier period.

Some cases are admitted in such a desperate condition that no one would recommend operation. I cannot honestly say that I have seen improvement follow operation in more than one case. Yet there is no alternative, and an insistence on this point may lead in course of time to a more general recognition of the necessity of early operation in such cases, so that patients showing diffused appendicular peritonitis may be at once placed under surgical care.

(c) So-called relapsing perityphlitis.

It must be borne in mind that a "relapsing perityphlitis" is in no way a special form of the disease. But we meet with a certain number of patients in whom attack follows attack with scarcely any interval of quiescence, so that the victim may pass a year or more in which he is unfitted for any active work.

The following is an example of the kind of case with which one is frequently called upon to deal.

CASE XV

H. M. H., aged 26, was seized at the end of February by a sudden pain in the right iliac fossa. This was attributed to a strain. He remained in bed for one day only, and then considered himself well again.

A second attack occurred three weeks later, in the middle of March. He was seized at midnight by a pain in the epigastrium, which rapidly increased in severity and spread down each side of the abdomen. A few hours later he began to vomit, while the pain began to settle down in the right lower quadrant of the abdomen. The vomiting was unusually severe, and he was sick no less than thirty-five times. On the next day he was found to have slight fever: there was resistance over the right side of the abdomen, and a soft ill-defined mass could be felt in the right iliac fossa. He remained under treatment for ten days.

A third attack occurred after an interval of three weeks, and this was similar to that previously described.

A fourth attack brought him again under my care on May 20. This was of the same character, but of less severity.

Thus four definite attacks had occurred in the space of three months.

On May 31 Mr. Clutton excised the appendix, which is described and shown in Figs. 21 and 22.

In this case I believe it may be said that the patient would almost certainly have had subsequent attacks, if his appendix had not been excised. And it is not unlikely, if the cystic condition had been developed (an event which the microscopical examination already described renders probable), that his life would have been endangered.

Such an instance as this, where four attacks occur in three months, and at the end of that time all further trouble and anxiety are removed by excision of the appendix, may be placed in contrast with the following common type of disease.

CASE XVI

W. H. C. S., aged 25, being in perfect health up to this time, was seized at the end of 1888 with a fairly severe attack of simple perityphlitis, from which he recovered completely. In 1889 he experienced at least three similar attacks, and in 1890 four more. In 1891 he had two attacks, but these were of a slighter nature. All these attacks were characterised by the same symptoms: the fever usually lasted three or four days, he was out of bed at the end of a week, and well again at the end of a fortnight.

He has remained in good health since but for occasional slight symptoms in the right groin pointing to impeded peristalsis, and he may now be considered to be cured.

Now such cases as these, under the preventive method of treatment advocated above, would never be seen again. But until the appendix is excised as a routine practice after a first attack, the question of dealing with this appendix-ridden class must be faced.

All the credit of initiating in this country a rational treatment of this condition lies, as already mentioned, with Treves. But his latest publication[70] on the subject has a less decided tone. "In deciding upon this operation (excision of the appendix in relapsing perityphlitis) it must be borne in mind that the measure is only justifiable in properly selected cases." All will agree that it is not wise to recommend operation in every case without discrimination. But the ground on which discrimination is made should be the question of the possibility of separating the appendix from its adhesions. There should be no question of the possibility of a mistake in diagnosis. And the statement that "there are a series of cases of relapsing typhlitis which do not depend upon appendicular trouble and which would not be relieved by the resection of the process" is, I believe, against the evidence of all recorded facts.

I do not believe that any hard rule can be laid down in these cases as to when to recommend operation and when to counsel patience. But I believe there are very few instances in which an operation should not be attempted. And it must be remembered that every succeeding attack renders the operation more difficult.

The two points on which a decision should be founded in any individual case are—(1) the position of the patient as regards the necessity of earning a livelihood, and (2) the number of previous attacks.

Operation in these cases must be regarded as directed to a saving of time rather than of life. The operation must not be allowed to involve a risk to life, because the disease itself entails scarcely any such risk. The first requisite, therefore, is a surgeon who is strong enough to recognise the impossible. The greater the number of previous attacks, the nearer does the impossible approach; and it must be freely confessed that in some cases of the quickly relapsing form the appendix cannot be safely excised owing to the density of the adhesions. Two cases have been under my care, in both of which the operation was wisely left incomplete. In one case the appendix was bound down to the posterior surface of the cæcum, and in the other it was firmly adherent and partly concealed in the ileo-cæcal fossa.

While it is recognised, on the one hand, that this relapsing form has scarcely any tendency to death, and, on the other hand, that in some cases excision of the appendix is not possible with safety, I think that few cases occur in the wage-earning class upon whom this operation should not be strongly urged.

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THE END.

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